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A MANUAL OF DISEASES OF THE STOMACH

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A MANUAL OF DISEASES OF THE STOMACH

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BY

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TORONTO LIBRARY OF MEDICINE

To

HIS FORMER STUDENTS

OF THE UNIVERSITY AND WESTERN INFIRMARY,

GLASGOW,

THIS BOOK IS DEDICATED

BY THE

AUTHOR.

PREFACE.

I have so often been asked by my old students to put into book form what I have tried to teach at the bedside and in the clinical laboratory of the diagnosis and treatment of Diseases of the Stomach that I have attempted in this volume to meet their desire by writing a short practical manual, which I trust the practitioner will find a useful introduction to the study of gastric diseases.

It requires, perhaps, some courage to write a new book on this subject, for so considerable a number have recently been published that it seems superfluous to add to the list. But, for the most part, recent works on the diseases of stomach have either been large and chiefly adapted for the specialist or they have been too incomplete and theoretic to be very useful to the medical practitioner.

This work aims at the middle course of being suitable for the student and practitioner, and it has at least the merit of being based almost entirely on personal experience and observation of gastric cases in my hospital and private practice. I have, however, on this account not wholly neglected to refer to the views and investigations of recent authors, and I have consulted the more important works on this subject as well as the articles which have appeared in the current European and American journals.

Long experience as a teacher and examiner makes me sure that there is no subject which more requires to be presented clearly to the practitioner than that of Diseases of the Stomach. I have therefore tried, at not too great a length, to present the subjects dealt with in a simple and practical manner in the hope that all who desire to become more familiar with the diagnostic methods of gastric affections will find what they require in these pages.

I have placed at the end of this book a Glossary giving, in outline, the laboratory apparati, reagents, and tests referred to in the text. There is nothing in the various investigations beyond the acquisition of any practitioner. A little practice will enable him to become

familiar with the reactions, and the time spent in these simple researches will be amply repaid by a more confident diagnosis and by indications for treatment.

I am much indebted to my friend, Dr. James R. Riddell, Lecturer on Radiology and Electro-therapeutics, Glasgow University, and Radiologist, Western Infirmary, Glasgow, for the admirable skiagrams illustrating the chapter on Radiography; and to my assistant, Dr. J. Salisbury Craig, for drawing the illustrations which appear throughout the book.

I have to acknowledge, with thanks, the kind permission of Messrs. P. Blakiston's Son and Co., Publishers, Philadelphia, to use the diagram of the Abdominal Viscera and Lungs (Plate cccxlvii, pp. 112-115), and the tabulated scheme of contents of the anatomical regions of the abdomen from Deaver's Surgical Anatomy, Volume III, which admirably illustrate the subjects to which they refer.

Wm. MacLennan.

4, Woodlands Terrace,
Glasgow, W.,
October, 1921.

CONTENTS

SECTION I.

ANATOMY, PHYSIOLOGY AND CHEMISTRY: CLINICAL AND LABORATORY METHODS OF EXAMINATION.

PAGE

INTRODUCTION 1

CHAPTER I.

ANATOMICAL CONSIDERATIONS.—The Stomach. Its Position and Structure. The Mucous Membrane and its Secretory Glands. Vascular Supply, Lymphatics and Innervation 7

CHAPTER II.

THE PHYSIOLOGY AND CHEMISTRY OF DIGESTION.—Insalivation, Salivary Digestion. Gastric Juice: HCl, proenzymes and enzymes. Organic Acids. Biliary Secretion. Pancreatic Secretion. Succus Entericus. Intestinal Micro-Organisms. Absorption. Motor Function 15

CHAPTER III.

ANAMNESIS.—Examination of the Patient. Gastro-Intestinal History Record. Significance of Symptoms and Signs 27

CHAPTER IV.

DISEASES OF THE ORAL CAVITY.—Parasitic Stomatitis. Aphthous Stomatitis. Ulcertive Stomatitis. Gangrenous Stomatitis. Pharyngitis. Treatment of Oral Affections. The Salivary Glands. Disorders of Salivary Secretion 45

CHAPTER V.

THE ŒSOPHAGUS.—Anatomy and Relationships. Abnormalities: Diverticula, Deformities. Examination. Diseases: Inflammation, Ulcers, Foreign Bodies, Spasm, Simple and Malignant Stenosis. Treatment 50

CHAPTER VI.

PHYSICAL EXAMINATION OF THE ABDOMEN.—Fat. Tympanitis. Ascites. Tumours. Enteroptosis 62

CHAPTER VII.

TOPOGRAPHIC RELATIONS OF ABDOMINAL ORGANS.—Liver, Spleen. Kidney (Movable Kidney). Differential Diagnosis of Kidney and Spleen. Pancreas 70

PAGE

CHAPTER VIII.

EXAMINATION OF THE STOMACH BY CLINICAL METHODS.—Physical Diagnosis: Inspection, Palpation, Percussion, Auscultation, Auscultatory Percussion. Examination by Inflation. Gastroscopy. Transillumination. X-Rays 82

CHAPTER IX.

RADIOGRAPHY.—Preparation of the Patient. Investigation of Œsophagus, Stomach, Intestines, Urinary Apparatus, Biliary Apparatus. The Duodenal Cap. Duodenal Ulcer. Dilatation of the Stomach. Gastroptosis. Hour-glass Stomach 90

CHAPTER X.

GASTRIC LAVAGE.—Technique and Apparatus. Use of Antiseptics. The Gastric Douche. The Gastric Spray. The Powder Insufflator 98

CHAPTER XI.

EXAMINATION OF THE SALIVA AND VOMITUS.—Macroscopic, Microscopic, Chemical, Bacteriological 104

CHAPTER XII.

TEST MEALS.—(A) Technique. Stomach Tubes. Expression. Extraction. (B) Examination of the Extracted Meal. Macroscopic Appearances. Qualitative and Quantitative Tests for HCl, Total Acidity, Combined Acid. Examination for Organic Acids. Macroscopic and Chemical Tests for Blood (Occult, Guaiac, Aloin, Teichmann's Hæmatin, Benzidine). Spectroscopic Analysis. (C) The Soluble Ferments. (D) Microscopic Examination of the Test Meal 109

CHAPTER XIII.

EXAMINATION OF THE FÆCES.—The Schmidt and Gaultier Diets. Macroscopic Examination. Chemical Examination. Microscopic Examination 143

CHAPTER XIV.

MOTOR POWER AND ABSORPTIVE CAPACITY OF THE STOMACH. Tests 155

CHAPTER XV.

DIETETIC PRINCIPLES.—General Considerations. Caloric Values in relation to Meals. The Mixed Meal. Eggs and Milk. Composition of Foods in common use. Vitamins. Digestibility of Foods 158

CHAPTER XVI.

RECTAL ALIMENTATION.—Indications for its employment. Simple *versus* "Nutrient" Enemata. Technique 167

SECTION II.

ORGANIC DISEASES OF THE STOMACH.

PAGE

CLASSIFICATION OF DISEASES AND DISORDERS OF THE STOMACH 171

CHAPTER XVII.

GASTRITIS.—Acute Catarrhal Gastritis. Phlegmonous Gastritis. Toxic Gastritis. Acute Infectious Gastritis. Chronic Gastritis (Simple, Acid, Hypertrophic and Sclerosing, Atrophic) 173

CHAPTER XVIII.

PEPTIC ULCER (ULCUS VENTRICULI: ROUND ULCER).—Etiology and Pathology. Classical Symptoms. Prognosis and Treatment. Evolution and Complications 196

CHAPTER XIX.

NEOPLASMS OF THE STOMACH.—Cancer of the Stomach. Symptoms and Signs. Presence of Tumour. Specific tests for Gastric Carcinoma. Special Features of Malignant Pyloric Stenosis. Treatment 233

CHAPTER XX.

DEFORMITIES AND DISPLACEMENTS OF THE STOMACH.—True Obstructive Dilatation (Simple Pyloric Stenosis). Hour-glass Contraction of the Stomach. Gastroptosis. Congenital Stenosis of the Pylorus 264

SECTION III.

FUNCTIONAL DISEASES OF THE STOMACH.

CHAPTER XXI.

DYNAMIC DISORDERS.—Disorders of Secretion (Hyperchlorhydria, Gastro-succorrhœa). Achylia. Heterochylia. Amyxorrhœa Gastrica. Myxorrhœa Gastrica 281

CHAPTER XXII.

DISORDERS OF MOTOR FUNCTION.—Myasthenia Gastrica. Acute Gastric Dilatation. Chronic Gastric Dilatation. Cardiospasm. Pylorospasm. Peristaltic Unrest. Aerophagy. Pneumatosis. Merycismus. Regurgitation 300

CHAPTER XXIII.

SENSORY DISORDERS.—Neurasthenia Gastrica. Hyperæsthesia Gastrica. Gastralgia Nervosa. Bulimia. Polyphagia. Sitisphobia. Parorexia. Anorexia Nervosa 318

SECTION IV.

STOMACH SYMPTOMS IN RELATION TO DISEASES OF OTHER ORGANS.

PAGE

CHAPTER XXIV.

DISEASES OF PANCREAS, GALL BLADDER AND BOWEL.—Diseases of the Pancreas. Cholelithiasis. Cholecystitis. Intestinal Disorders (Obstructions, Appendicitis) 331

CHAPTER XXV.

DYSPEPSIAS OF PULMONARY, CARDIAC, TOXIC AND BLOOD DISEASES.—The Dyspepsias of Pulmonary Affections (Phthisis, Chronic Bronchitis, Emphysema, Asthma). Cardiac Disease. Chronic Nephritis. Diabetes Mellitus. The Dyspepsia of Anæmia 342

CHAPTER XXVI.

VOMITING AS A SYMPTOM.—Persistent Vomiting. Bilious Vomiting. Nervous Vomiting. The Vomiting of Pregnancy .. 347

CHAPTER XXVII.

CONSTIPATION.—(Chronic, Atonic, and Spastic). Causes. Treatment 352

CHAPTER XXVIII.

DIARRHŒA.—Causes. Varieties. Treatment 364

GLOSSARY OF TESTS 371

INDEX 381

LIST OF ILLUSTRATIONS.

PLATES.

Plate I.	The Cæliac Axis Artery and its branches ..	*facing page*	12
„ II.	Skiagrams of normal and abnormal stomachs ..	„ „	96
„ III.	„ „ „ „ „ „ ..	„ „	96
„ IV.	„ „ „ „ „ „ ..	„ „	96

IN THE TEXT.

FIGS.		PAGE
1, 2, and 3.	Position and Form of empty, half-filled and full Stomach ..	9
4, 5, and 6.	Longitudinal, Circular and Oblique Fibres of the Stomach ..	10
7.	Cardiac Gland	11
8.	Pyloric Gland	11
9.	Tubule of a Fundus Gland of Human Stomach..	12
10.	Capillaries of Gastric Gland of Rabbit	13
11.	The Lymphatics of the Stomach	13
12.	Diagram showing Relations of Œsophagus	50
13.	Einhorn's Divisible Œsophageal Bougie and Olives	55
14.	Diagram of Abdominal Viscera and Lungs, Anterior View ..	72
15.	Diagram illustrating methods of Auscultatory Percussion of Stomach	86
	Radiograms of normal and abnormal Stomachs ..	94 and 95
16.	Stomach Tubes: A. Ewald's tube. B. Boas' tube. C. English pattern	111
17.	Kuttner's Extractor	113
18.	Weiss Tube	123
19.	Strauss Separating Funnel	125
20.	Teichmann's Iodo-Hæmatin Crystals	131
21.	Spectra of Oxyhæmoglobin and various Derivatives.. ..	133
22.	Objects normally found in Gastric Contents	138
23.	Leptothrix Buccalis and Yeast Cells	140
24.	Sarcinæ Ventriculi (simple pyloric stenosis)	140
25.	Boas-Oppler Bacilli and Yeast Cells	140
26.	Strassburger's and Schmidt's Fermentation Tube	148
27.	Fat Droplets. Fat Crystals. Vegetable Tissue. Starch ..	150
28.	Slide of Fæces	152
29.	Abnormal Fæces	153
30.	Abdominal Outlines of Gastroptosis in erect and recumbent postures	275
31.	Diagrammatic representation of method of performing auto-abdominal massage	355

INTRODUCTION.

There are many reasons why diseases of the stomach are comparatively neglected by the general practitioner.

First, our students leave hospital with a smaller amount of knowledge of gastric affections than of any other system.

Secondly, diseases of the gastro-intestinal system, like abdominal conditions generally, are admittedly very difficult of diagnosis by the ordinary methods of examination.

Again, symptoms of gastric disturbance are frequently common to many disorders of wholly different origin.

To unravel and allocate aright the evidences of disturbed digestive function requires clinical experience, and often a laborious investigation and a knowledge of certain laboratory methods which the overburdened student has had no time, or opportunity, of acquiring before leaving our clinics. It is lamentable that this should be the case, because almost certainly he will be called upon to deal with digestive disorders more frequently than with any other ailments.

The stomach is the only organ over which we have almost complete control, and, perhaps for this very reason, it is the 'best abused' organ in the body.

Every age has its fashions in medical practice, and at the present time we are living in the surgical era. The comparative safety, so far as life is concerned, with which the abdomen can now be opened has had an important influence in promoting accuracy in the diagnosis of diseases of the stomach. Because, no doubt, we have learned much from direct surgical observation, I am not prepared to transfer this department of medicine to the domain of pure surgery. Experience of a large number of post-operative cases constrains me to say that surgical treatment is ultimately less frequently successful in bringing relief to the patient than many surgeons imagine.

In making this statement it is far from my intention to belittle the indisputable value of operation in suitable cases. The tendency

to-day, however, is to expect too much from surgery, and needlessly to submit cases that are amenable to medical treatment to surgical interference.

I do not believe that laparotomy can be performed without the possibility of formation of some adhesions which may, and frequently do, give rise to troublesome sequelæ. After gastro-enterostomy, as long as the patient remains in hospital, symptoms are completely in abeyance, but, later on, when the strain of life recommences, general and local symptoms may supervene. Such patients often become neurasthenic, or they may suffer from bilious regurgitation and permanently remain in a state of indifferent or even miserable health. On the other hand, from want of an appreciation of the nature of the disease, stomach cases which ought to be at once handed over to the surgeon are kept on medical treatment till any hope of successful operation has passed. Many a useful life might be saved if the practitioner had a sounder knowledge of gastric lesions enabling him to estimate when a case ceased to be medical and became surgical.

I do not claim that in every instance, even after the most complete investigation by clinical and laboratory methods, a correct and exact diagnosis can be made. But if, at times, only an approximate diagnosis can be obtained, after enlisting every method of investigation at our disposal, how much less certainly is the surgeon entitled to give an opinion as to the necessity for operation without invoking the aid afforded by these researches ?

It is because the significance of the early symptoms of gastric diseases are so little comprehended that so many become surgical affections. I am of opinion that peptic ulcer (gastric and duodenal) is still a medical ailment, and that if the early symptoms and signs were better appreciated they would never need to become surgical diseases. There are, of course, exceptions to this general rule, as when violent hæmorrhage or perforation are the events which intimate the presence of disease.

The first essential in any clinical investigation is a thorough and complete examination of the patient. Of no disorders is this more true than those of the digestive tract.

A general examination should first be undertaken. In a great many cases a group of symptoms, apparently referable to the digestive organs, may have its origin in other systems altogether. It is not uncommon to have symptoms of digestive disorder from

certain renal and cardiac affections, or they may be produced reflexly from disease in other organs. It is therefore necessary to exclude these before an investigation of the gastric condition is undertaken.

A careful examination of the heart, lungs, kidneys, and blood should be made at the outset, or the dyspepsia of phthisis, cardiac disease, renal disorder, or pernicious anæmia may be wrongly ascribed to a primary gastric affection. Too frequently, in digestive derangements, the abdomen is not systematically examined. The physician is apt to be content to treat some of the prominent symptoms of dyspepsia by a modified diet and some medicinal agents. No wonder that patients suffering from diseases of the digestive system fall into the hands of quacks who find in the dyspeptic an enormous clientèle.

During the last decade great progress has been made in gastro-intestinal pathology. This is largely due to laboratory investigation and to advance in physiological chemistry. But we owe the largest amount of progress in the elucidation of digestive derangements to direct interrogation of the work going on in the digestive tube by means of the stomach tube, X-ray investigation, and surgical observation. It is not always true that one can utilise the results of laboratory work at the bedside, and experimental observation of the digestive functions of the lower animals does not always yield reliable data on which to base treatment of disease in the human subject.

There is a decided tendency to-day to belittle the results obtained by an investigation of the gastro-intestinal chemistry. I believe this attitude arises from insufficient experience. The information derived from the use of test meals is of the utmost importance for diagnosis and treatment, and no vindication of this method of investigation is at all called for.

It is, however, perfectly true that no study of the gastro-intestinal chemistry *alone* will ever lead to accuracy of diagnosis, but I do maintain that, when conjoined to other methods of investigation, it is one of the most important in determining the nature of the disease and in differentiating functional from organic, simple from malignant diseases.

Unfortunately all organs cannot be directly interrogated. It will be admitted that an examination of the secretions or excretions of an organ usually affords valuable information of its function

and of the presence or absence of disease. We are almost wholly dependent on an examination of the urine for the diagnosis of renal disorders. Although certain kidney diseases produce definite clinical pictures, the physician who made a diagnosis without reference to the urine would certainly frequently fall into serious error. So in the case of gastric diseases the method of direct interrogation, by means of the stomach tube, is equally important and should never be neglected.

Because, in different individuals, wide variations in the chemistry of the test meal are found in the same diseases, some physicians and certain surgeons hastily discount this method of investigation. The whole process, from the passing of the tube to the macroscopic, microscopic, and chemical investigation must be carefully considered and the information gleaned from these studies must be co-related to the symptoms and signs. When one has acquired sufficient experience, valuable information of the secretory and motor functions of the stomach may be obtained by this examination. A consideration of the general appearances of the extracted meal, of its amount, colour, smell, degree of digestion, reactions, chemical findings and microscopical appearances yields information that is indispensable for diagnosis and treatment.

When pepsin was discovered to be the active enzyme of the stomach, it held for long the therapeutic field in the treatment of all kinds of gastric derangements. But the truth is that in the treatment of most gastric ailments it gives us little help and seems to play a far less distinguished rôle than it was credited with some years ago. To a less extent the same may be true about hydrochloric acid, which activates the pepsinogen. If the pancreatic and intestinal digestive agents remain active the stomach digestion can, in many instances, be largely dispensed with, at least for a time.

Our range of therapeutic applications has enormously increased, and we can now bring to bear upon diseases of the alimentary tract a great variety of influences. It is quite true that some surgeons claim that most of the organic diseases of the stomach are no longer medical and that their proper treatment is surgical. Because the abdomen can be opened nowadays with comparative safety, so far as the patient's life is concerned, it does not follow that the best practice is to hand over all such cases to the surgeon's care.

The classification of gastric diseases is more or less arbitrary. Most writers adopt a pathological basis, but this is not, and cannot be, strictly adhered to. Many, indeed, about three-fourths of all gastric ailments, are associated with recognisable pathological change, but in all our classifications we have to incorporate ailments based on abnormal function. Thus we are able to adopt a pathological classification of such diseases as peptic ulcer and gastric carcinoma, but we cannot utilise it for such ailments as hyperchlorhydria, benign achylia, and certain neuroses.

As a matter of fact, the classification of gastric disorders is of secondary importance. For the clinician the all-important thing is the recognition and appreciation of disordered function. What is essential is a sound knowledge of the normal functions, complex as they are, of the stomach, and a capacity to ascertain, with more or less certainty, where the error is, how it has been produced, and what treatment is required to remove it.

To make a correct diagnosis of the cause of a dyspepsia entails much experience and often a tedious investigation. To attempt to diagnose and treat certain cases from the symptoms alone is almost sure to lead to error and to bring little credit to the practitioner. A sound opinion of the nature of certain digestive troubles can only be based on a careful history of the symptoms, a thorough examination of the physical condition of the digestive tract, and a complete study of the gastro-intestinal chemistry.

In *Radiography* we have also a potent diagnostic ally in obscure abdominal diseases. Too much must not be expected from this method of investigation, but when well carried out by an experienced radiologist most reliable diagnostic data may be obtained in suitable cases. But, just as a study of gastro-intestinal chemistry alone should never be relied on, so also, in the case of radiography, the behaviour and the appearances of the opaque meal divorced from symptoms and signs may be very misleading. The truth is that it is only by a combination of clinical and laboratory methods that we may hope to make a creditable diagnosis of many digestive troubles.

In the hope of making the student take a greater interest in diseases of the stomach, and to enable him and the practitioner to know how to investigate such cases aright, these pages are written.

SECTION I.

ANATOMY, PHYSIOLOGY AND CHEMISTRY: CLINICAL AND LABORATORY METHODS OF EXAMINATION.

CHAPTER I.

ANATOMICAL CONSIDERATIONS.

In a short practical manual of Digestive Derangements it will be impossible to give full anatomical details, but an adequate knowledge of the subject is so essential to the clinician that the student and practitioner who are interested in this department of work are referred to the standard works on Anatomy.

It will be only necessary to give an outline of the more important details of the anatomical relationships of the stomach and of its macroscopic and histological anatomy.

The Stomach.—The empty stomach lies in the left hypochondriac and epigastric regions. It is kept in position by the œsophagus and by certain folds of peritoneum, viz., the gastro-phrenic, gastro-splenic, gastro-hepatic, and gastro-colic omenta.

It is roughly divided into three parts: the cardiac and the pyloric portions, and between these two is the body of the stomach.

The part of the cardiac end projecting to the left of the œsophagus is the fundus (great *cul de sac*). The dilated part at the pyloric end is the pyloric antrum. The pylorus is usually demarcated by the venous ring which surrounds it.

A little to the left of the tenth dorsal vertebra lies the cardiac orifice, and about one inch to the right of the eleventh and twelfth

dorsal vertebræ the pylorus will be found. A line drawn between these two points indicates the normal position of the organ in its long axis.

For the physician the pylorus is one of the most important regions of the stomach. Below and behind it lies the pancreas, and above and in front the liver. At rest and during the first part of the digestive act it remains closed. It opens from time to time, and some of the chyme is passed through it into the duodenum. Later, when digestion is nearing completion, the contraction lessens and the orifice becomes capable of further distension.

The pylorus is very movable, and is found in different positions according to the condition of the stomach. When the viscus is empty, the pylorus lies about half an inch from the middle line beneath the left hepatic lobe, but when the stomach is full, the pylorus occupies a position beneath the quadrate lobe further still to the right.

The upper curved margin is the lesser curvature and the lower and longer border the greater curvature of the stomach.

Anatomical Relations of the Stomach.

In front are the diaphragm, abdominal wall, and liver.

Behind lie the spleen, pancreas, left kidney, left supra-renal capsule, transverse meso-colon, diaphragm, aorta, inferior vena cava, solar plexus and bodies of the vertebræ which indent the posterior wall of the stomach in the dorsal decubitus.

Above are the diaphragm, liver, and lesser omentum.

Below the stomach rests on, and is supported by, the top of the left kidney and gastric surface of the spleen, the top of the pancreas and the transverse meso-colon.

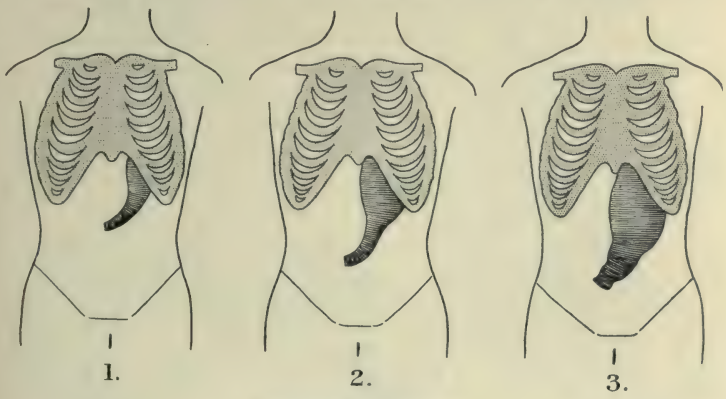
As many diseases of the stomach implicate other organs and parts in the neighbourhood, it is important to remember its relationship to these.

The stomach is not an inert sac, but an actively contractile organ. When the stomach receives food the fundus first expands and later, but to a less extent, the whole cardiac end distends. At this stage about four inches of the pyloric end remains contracted, but a little later it too becomes expanded, while a further general increase in size and greater obliquity of its long axis makes the great curvature come in contact with the abdominal wall in front.

It is important to note the different positions and forms taken by the stomach according to the amount it contains. Hertz has shown by X-rays and opaque meals that :—

The *empty stomach* is pear-shaped in its upper third and contains air. The remainder resembles a collapsed tube ending in the pylorus, which occupies a position in the mid-line between the upper margin of the symphysis pubis and the upper portion of the ensiform. The greater curvature comes down to about the umbilicus, in the vertical position, while in the horizontal the organ lies more obliquely and the greater curvature does not descend quite so far.

The *half-filled stomach* is shown by the opaque meal to be more uniformly distended, while its axis is that of the empty viscus. Now, however, in the erect posture, the greater curvature reaches below, and in the horizontal always above, the intercristal line. The pylorus rises or falls, to some extent, with the position assumed.



Position and Form of
(1) Empty Stomach. (2) Half-filled Stomach. (3) Full Stomach.

The *full stomach.* The left half of the diaphragm becomes pushed up so as to be as high as, or higher than, the plane of the right half. The diameter of the body of the stomach remains uniform, but all the dimensions are greatly enlarged and the pylorus itself is pushed a little to the right.

The stomach is thus liable to great alteration in position, according to the amount of gas or food it may contain. When distended it pushes the transverse colon and small intestine downwards

and the diaphragm, heart, and lungs upwards. Gastric distension may thus cause embarrassment of cardio-respiratory action.

The normal position of the stomach is liable to variation by alterations in the neighbouring organs—*e.g.*, pleurisy with effusion.

When the stomach is empty it retracts, and the space left in front is gradually filled by transverse colon. In physical examination of the stomach this is an important fact to bear in mind.

It is a very common mistake to diagnose succussion, originating in the colon, for a gastric splash.

Structure of the Stomach.—From without inwards we have :—

(1) Peritoneal covering.

(2) Muscular coat formed of unstripped fibre and consisting of three layers—

(*a*) External and longitudinal fibres connecting with the œsophageal and pyloric fibres. These are most abundant on the lesser curvature, and form a dense mass over the pylorus.

(*b*) A middle coat of circular fibres continuous with the superficial circular fibres of the œsophagus. They are least abundant over the lesser curvature, and most numerous, dense, and thick at the pylorus, and form the ' valve.'

(*c*) The internal fibres are arranged obliquely, principally on the fundus. These fibres are continuous with the deep circular fibres of the lower end of the œsophagus.

Fig. 4.
Longitudinal Fibres.

Fig. 5.
Circular Fibres.

Fig. 6.
Oblique Fibres.

(3) The sub-mucous coat, composed of loose consecutive tissue, is closely attached to the mucosa, and carries the blood vessels and nerves, which divide here before entering the mucous membrane.

(4) The mucous membrane is soft and pulpy. Its colour varies greatly according to its activity. Post-mortem it is greyish, but during life the depth of its pinkish colour varies with its vascularity.

It varies considerably in thickness in different situations. Thinnest at the fundus, it is comparatively thick over the pylorus. The rugæ which form so conspicuous an appearance are due to the fact that the mucous membrane is large enough to cover, without stretching, the stomach when fully distended.

The contraction of the outer coats when the viscus is empty throws the mucous membrane into folds or rugæ. The mucous membrane shows numerous round eminences called 'mammillæ.' In these are seen small apertures—the mouths of the gastric glands—which are packed more and more closely together as they

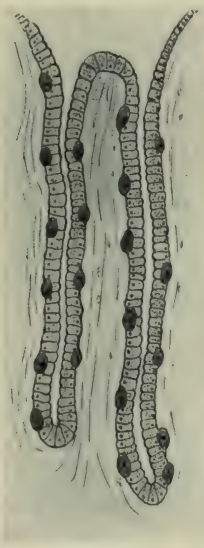


Fig. 7.
Cardiac Gland.

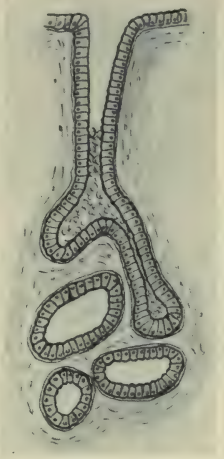


Fig. 8.
Pyloric Gland.

approach the pylorus. It has been estimated that there are no fewer than five millions of tubular glands in the stomach. These glands are covered by columnar epithelium which extends for some distance into their mouths, and are surrounded by fibrous tissue and lymphoid cells.

There are three varieties of glands found in the stomach :—

(1) *Cardiac Glands* are found in the cardia and fundus. The greater part of the stomach contains these very long glands with

short mouths. Numerous small granular columnar cells line the lumen of the glands, and larger parietal oval granular cells lie between these in smaller number. The parietal have a much greater affinity for aniline dyes than the columnar cells.

The pepsinogen and milk-curdling ferment are secreted by the columnar; the hydrochloric acid by the parietal cells.

(2) The *Pyloric Glands* differ materially from the others. They have long mouths lined by cylindrical epithelium. They contain only central cells, which secrete no acid, but only pro-ferments. These are found at the pylorus alone.

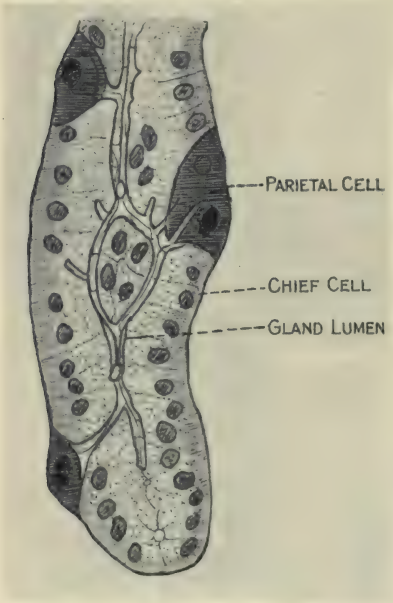


FIG. 9.
TUBULE OF A FUNDUS GLAND OF HUMAN STOMACH (after Zimmermann).

(3) *Mucous Glands.* These are found most abundantly where the mucous membrane shows most rugæ—that is, where it is thickest. The pyloric end of the stomach has thus fewer mucous glands than the cardiac, where the rugæ are most numerous.

The Vascular Supply of the Stomach.—The arterial supply is derived from the cœlic axis. At the lesser curvature the large coronary artery joins the stomach, gives off a branch to the œsophagus, and then divides into two portions, which run along the smaller curvature, one on either side. These join with two vessels from the pyloric branch of the hepatic artery, and they too run on the smaller curvature, one on either side. An arch is thus formed anteriorly and posteriorly, and from it large branches supply the anterior and posterior surfaces of the stomach, perforating the muscular coat at different situations.

The greater curvature is supplied from branches of the right and left gastro-epiploic arteries (branches of the gastro-duodenal

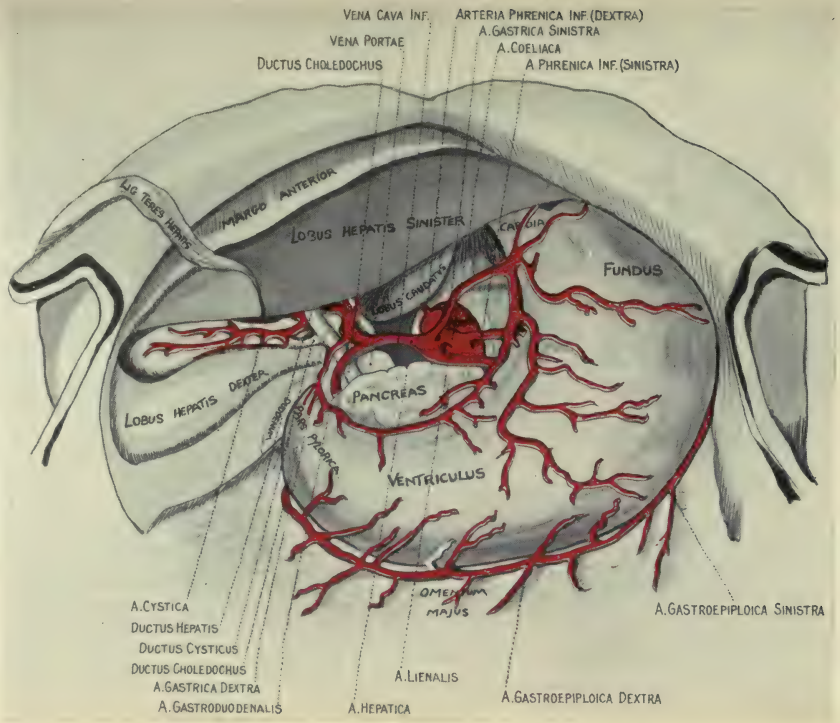


FIG. 10. THE CŒLIAC AXIS ARTERY AND ITS BRANCHES.

(After Kopsch)

and splenic). These are found in the omentum, close to its attachment in the stomach.

The fundus derives its supply from short branches (running in the gastro-splenic omentum) from the splenic artery.

All these blood vessels to the stomach break up after perforating the muscular coats, and form, in the mucous membrane, a capillary network surrounding the tubules. The vessels are so arranged that about two-thirds of the total arterial supply goes to the mucous membrane and its glands.

Fig. 10.
Capillaries of Gastric Gland of Rabbit (after Golgi).

The veins take origin from these capillaries and form larger trunks in the submucosa. From these trunks, by smaller vessels, the venous blood passes out through the muscular layer to form finally the coronary and pyloric veins, emptying into the portal vein. The right gastro-epiploic vein empties into the superior mesenteric vein, and the left into the splenic vein.

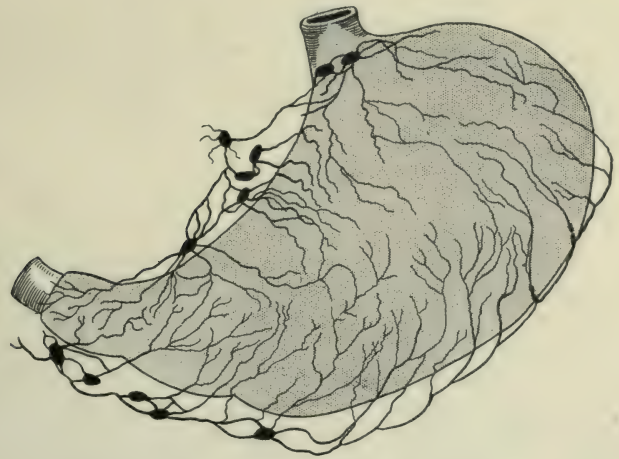


Fig. 11.
The Lymphatics of the Stomach (Moynihan after Cunes).

The Lymphatics correspond in their arrangements to the veins. The lymphatic glands extend along the lesser curvature as far as the cardia, and along the greater curvature from the pylorus, about one-third of its length. It is imperative to remember the situation of these glands in carcinoma of the stomach, especially if a radical operation be attempted.

The Innervation of the Stomach is mainly derived from the two vagi and solar plexus of the sympathetic system. The left vagus passes in front of the right behind the œsophagus. Both pierce the diaphragm along with the gullet, and pass to the upper and lower surfaces of the stomach respectively. Joining with the sympathetic filaments they form the cœlic plexus. In the submucous layer these nerves divide into numerous branches with local ganglia.

CHAPTER II.

THE PHYSIOLOGY AND CHEMISTRY OF DIGESTION.

It is not intended here to give a detailed account of the complex processes of digestion, but to outline only the more important steps in the preparation of aliments for absorption. For the most part these changes, involving the conversion of food into nutritive material fit for absorption, are due to the action of specific ferments found in saliva, gastric juice, pancreatic juice, and the intestinal secretions, aided by the influence of bile.

In considering the important functions of the gastro-intestinal organs, and the part they play in the digestive processes, it must be kept in mind that we are dealing with most complex and interdependent actions. Alimentary substances are subjected to so many changes—chemical and physical—during their passage through the digestive tract that it is well nigh impossible to ascribe a rôle of greater importance to one than to another. Disease, or disorder of one part, is commonly associated with symptoms of disorder in another. The difficulty of gastro-intestinal diagnosis is not only enhanced by this interdependence of function, but also by the presence of other and new symptoms, due to reflex action or to the presence of disease of abdominal viscera which have no direct connexion with the digestive organs themselves.

The first part of the digestive process begins in the mouth. The food is subjected to mastication and insalivation. During mastication the food is more or less thoroughly mixed with saliva and, through the action of ptyalin, is subjected to its initial digestive process. The amount of saliva secreted has a direct relationship to the length and thoroughness of mastication, but it is also influenced by the various reflexes—more particularly by the senses of taste, smell, and sight. The secretion of gastric juice is similarly affected by reflex action (psychic juice). It is thus that a

well-prepared and well-cooked meal, served in a dainty way, is not an unimportant stimulant of appetite and aid to the digestive process.

It is very important that the saliva should be kept free from contaminations derived from the mouth or the nasopharynx. Oral sepsis as a pathogenic factor in many gastro-intestinal and constitutional diseases must always be kept in mind.

The mixed saliva is normally alkaline in reaction. It is quite probable that this alkaline secretion acts as a stimulant of the gastric glands and promotes the flow of gastric juice.

The importance of thorough and methodical mastication as a preparatory act to digestion needs almost no comment. Not only does mastication ensure a fine state of division of the solids but it also evokes, mechanically and reflexly, an abundant secretion of the essential digestive fluids. Thus it is that food habitually hurriedly and insufficiently chewed sooner or later results in digestive derangements.

In abnormal conditions of health it not infrequently happens that the reaction of the mixed saliva may become changed from alkaline to acid. From an extensive series of observations I have found that this is not a very uncommon occurrence, especially in young gouty and rheumatic subjects. It is certain that when the saliva is continuously of an abnormal acid reaction it must exert a most harmful influence in promoting dental caries and lead ultimately to oral sepsis. An acid salivary secretion will also compromise carbohydrate digestion.

Ptyalin, the diastatic ferment of the saliva, begins in the mouth the conversion of starch into sugar or maltose. This process is continued in the stomach during the earlier stage of digestion, but is arrested when the gastric juice gathers sufficiently in acidity and amount to neutralise the alkalinity of the saliva swallowed. The starchy content of the diet remains undisturbed in the cardiac end, where the ptyalin maintains its activity till inhibited by the acid of the gastric juice. Many serious functional derangements, often included in the term of "Amylaceous Dyspepsia," are due to a premature arrest of the further digestion of starches in the stomach by excess of hydrochloric acid. In all cases of gastro-succorrhœa and hyperchlorhydria this type of dyspepsia is common.

Maxwell has shown that ptyalin has also a hydrolysing influence on starch and thus helps gastric digestion.

The Gastric Juice.—As in the case of the salivary, so also for the gastric secretion, many reflex and other actions come into play as stimulants. Taste, smell, sight, and expectation all play a rôle. The mere thought of a savoury meal, particularly to a hungry man, will cause a flow of salivary and, probably, of gastric secretion.

The character and the amount of the food have also important influences. It has more recently been asserted that the salivary glands of dogs secrete a material which, when absorbed from the stomach into the circulation, has a stimulant action on the secretory glands of the stomach.

The active gastric secretion is a clear, colourless, or slightly yellowish liquid of acid reaction. The amount of acid varies greatly within normal limits—namely, from 0.1 to 0.4 per cent.

The important work of Pawlow has demonstrated the perfect adjustment which exists, in health, between the quantity and quality of the gastric and other secretions to meet the requirements of the food taken and to ensure its digestion. Idiosyncrasy and the nature of the food are important factors in determining the amount of acid and pepsinogen secreted.

Hydrochloric Acid plays many rôles in the digestive functions :—

(1) It is the first line of defence of the gastro-intestinal tract against infection from the outside. Without it many bacterial poisons taken with our aliment might become pathogenic. It is very powerfully antiseptic. In health, however, it is not present in the quiescent states of the stomach. The effect of the absence of hydrochloric acid is well seen in malignant disease. It permits the undigested and unacidified chyme to pass into the intestine in an ill-prepared or decomposing condition, and so promotes intestinal putrefaction. This putrefaction is, however, not wholly due to the mere absence of the free hydrochloric acid, for in simple achylia there may be little or no putrefaction either in the stomach or in the intestine.

Observation of many cases makes me feel sure that the degree of gastric acidity can be materially influenced by the intake of common salt—a point worth remembering in cases of obstinate hyperchlorhydria and gastro-succorrhœa. An excess tends to overproduction of hydrochloric acid. On the other hand, voluntary abstinence from salt will frequently produce achylia.

As the blood is alkaline the formation of hydrochloric acid can only be explained by a kind of selective action of the parietal cells.

It is probably elaborated from the chlorine of the common salt so abundantly present in the fluids of the body. It should be observed that the percentage of free hydrochloric acid varies greatly in apparently healthy individuals, and that the kind of food largely determines the quantity secreted. In dogs the percentage is very high, amounting to 0.4. Variation, too, may occur from the neutralising action of the alkaline saliva, and from the alkaline mucus. In a large series of investigations I have rarely found more than .3 per cent. of free hydrochloric acid without symptoms of disturbance, but some authors claim that it may be present in a still greater proportion without producing any abnormality.

(2) Hydrochloric acid would appear to influence gastric motility. There is commonly a distinct relationship between the amount of motor activity and the amount of acid present. But it is to be observed that in many cases of simple achylia there appears to be an abnormal degree of motility, and a very rapid emptying of the stomach after a meal. It may be that in these cases the less liquid condition of the chyme (from absence or diminution of the gastric secretion) may act as a stronger mechanical stimulant to the stomach, and so induce more active peristalsis. In addition, the absence of acid permits of a relaxation of the pylorus and contributes to the rapidity with which the stomach empties. (See Achylia Gastrica.)

(3) The pro-enzymes—pepsinogen and rennin-zymogen—are activated by the action of hydrochloric acid, and become pepsin and rennet. It is, however, true that these pro-enzymes may also be activated by other acids, as, for example, by lactic acid.

Hydrochloric acid has the power of combining with proteid material to form acid albumen or syntonin, and, along with pepsin, aids in the formation of peptone. It also has the power of converting cane into grape sugar.

(4) Acids as a rule stimulate glands, whose secretions are alkaline, and diminish the activity of glands whose secretions are acid. Thus the amount of acidity in the chyme, which passes into the intestine, will largely determine the amount of the secretion of the alkaline pancreatic and intestinal juices.

More recently speculation has centred round the action of the highly specialised glands in the upper part of the duodenum. It is assumed that they elaborate pro-secretin largely through the stimulant action of the acid chyme, and that the pro-secretin

forms, with the hydrochloric acid, secretin. This secretin when absorbed into the circulation is supposed to be the real stimulant of the pancreas. If this be so, the importance of a sufficient amount of acid in the chyme will be readily understood. It will also explain to a large extent how chronic duodenal ulceration, with a permanent hyperchlorhydria, is not uncommonly associated with a form of interstitial pancreatitis, due to continuous over-stimulation of that organ. Thus we find chronic peptic, especially chronic duodenal ulcer, with its tendency to supersecretion, not uncommonly associated with induration of the pancreas. The more common explanation of the effect of the acid chyme on the pancreatic secretion is that it is reflex.

It has been computed that about three pints of gastric juice are secreted in twenty-four hours. It is stimulated by the mechanical as well as by the chemical action of food. Water, dextrin, pepper and highly spiced foods, and most of all meat extractives, have a powerful peptogenic influence, while oils and fats depress the secretion. By its action albuminates are converted into soluble pro-peptones and peptones. Pepsinogen may be secreted even when no acid is present, but it is only active in an acid medium Gelatine loses its power of gelatinizing, and is converted into gelatine peptone. The rennin curdles the milk. Its action appears to be limited to this, and it is activated by the calcium salts. In the absence of these, rennin-zymogen is as inactive as is pepsinogen in the absence of hydrochloric acid. Rennin has not been isolated, and many observers deny its existence.

Probably a fat-splitting enzyme—*lipase*—is also present in the gastric secretion. It is active not only in the presence of acid, but also in neutral media. It has a powerful action on emulsified fats.

The stomach is more a preparatory than an absorptive organ. Water passes rapidly through it with little or no absorption. Only small amounts of the soluble materials—peptone, sugar and salts—may be taken up by the absorbent channels of the stomach. The bulk of the chyme passes through the pylorus to the intestine, where the more important digestive processes are performed, and where the real absorption takes place.

To the chemical processes going on in the stomach have to be added the physical. The amount of time required for digestion must vary greatly in different individuals, and must have a relationship to the size and character of the meal. The stomach empties

itself normally after an ordinary mixed meal, in from four to seven hours—a small taking the shorter, and a large meal the longer, period. The chyme escapes at intervals through the pylorus, which relaxes from time to time. The food is really expressed into the intestine by the peristaltic contractions of the viscus. The peristaltic waves are less active in the fundal part of the stomach, where food may remain undisturbed for some time.

Much work has recently been done to elucidate the character of the gastric movements by means of opaque meals and the X-rays. It has been shown that the movements start in the middle of the stomach and pass towards the outlet at the pylorus, and that these movements have a regular rhythm. The movements become more active and ample the more complete digestion may be. By relaxation and contraction of the pyloric end a certain amount of chyme is expressed into the bowel.

Many serious functional and organic troubles may arise from disorder or defect of the gastric motility, but these will fall to be discussed later.

The exact consistence of the chyme may exert a more potent influence on gastric activity than is usually supposed. Theoretically large volumes of water taken with solids might conceivably delay digestion, not only by diluting the gastric juice, but by separating the food from intimate contact with the walls of the stomach at the pyloric end. Nature prevents, however, any great dilution of the gastric juice and any prolonged separation of food from the stomach walls, by the rapidity with which fluids are thrown out of the stomach into the intestine.

The carbohydrates appear to pass most quickly, as they require only about half of the time that nitrogenous foods do for their gastric digestion. One of the main factors in promoting pyloric relaxation is the action of free HCl. As starches do not combine with HCl we find that a purely carbohydrate meal, such as a test breakfast, leaves the stomach quite rapidly, owing to the accumulation of the acid-charged chyme at the pyloric end of the stomach. It must be kept in mind that a similar relaxation of the pylorus occurs in simple achylia gastrica. This subject is discussed in the chapter on achylia. But the exact speed with which a meal leaves the stomach depends also on the manner in which it is taken. The ingested food appears to arrange itself in layers, or strata, in the stomach. Thus, if proteids be taken first, they accumulate

at the pyloric end and apparently delay the expulsion of the carbohydrates into the intestine.

In addition to these specialised enzymes and hydrochloric acid, the mucosa secretes mucus. It is most abundantly found in the cardiac end and less in the pyloric. It should be noted that often a considerable proportion of mucus returned in test meal or in vomitus may consist of what is swallowed. It then lies in flocculent masses separate from the meal. Mucus is digested by the gastric juice and ultimately becomes intimately mixed with the chyme.

When examining mucus microscopically, important information as to its source of origin may be obtained from the character of the cells mixed with it—especially when it is taken from the empty stomach. It is alkaline in reaction, and is a very important protection to the mucous membrane which secretes it. After a test meal it is seen partly mixed and partly free. In health it should be scanty, and, generally speaking, when there is abundant hydrochloric acid, it is small in amount, as in gastric ulceration. In malignant achylia and chronic gastritis it is abundant.

Fermentation in the stomach may be said to be normal or abnormal. It very largely depends on the degree in which it takes place whether we regard it as an incident of normal digestion or a sign of disorder of function.

An organ like the stomach, in free connexion with the outside world, is liable to all kinds of bacterial infection. The stomach may be a good incubating chamber during the periods it is not protected by the presence of a sufficient amount of antiseptic hydrochloric acid. But even a high percentage of HCl does not, under all conditions, exert a complete inhibitory influence on the development of micro-organisms.

Normally, organisms associated with fermentation are negligible. They only assume a rôle of importance when there is serious disorder of motility. If the motor function and tonicity of the stomach are good, gas, swallowed air, or gas resulting from fermentation, is eructated or passed into the intestine.

As almost all bread stuffs contain some active yeast cells, these act on the carbohydrates with the formation of gas, which tends to accumulate in the stomach. Such fermentation, when slight, may be regarded as an accompaniment of normal digestion. Yeast also splits up glucose and forms alcohol and carbonic acid gas.

Acetic acid fermentation due to Mycodermia Aceti acts on alcohol and forms acetic acid. This fermentation occurs chiefly in those who take alcoholic beverages with their meals, although acetic acid may also be formed from the action of yeast on starch.

But many fermentations are obviously abnormal, because so excessive. These are almost always associated with stagnation and retention of the gastric contents. Thus in the retention associated with malignant disease the lactic acid bacillus may be found swarming in the vomitus and producing much fermentation. The lactic acid bacillus acts on the lactose and produces lactic, acetic, and carbonic acids.

Various anærobic bacteria—*e.g.*, B. Butyricus—cause *butyric acid fermentation.*

In retention from simple pyloric stenosis, with high HCl acidity, we often find the stomach contents resembling a pure culture of Sarcinæ Ventriculi, and at times a large amount of yeast. In such cases an abundant gas-formation results from the fermentation set up by these organisms.

Although this manual is primarily concerned with gastric diseases, it is, as has already been indicated, impossible sharply to dissociate them from certain disorders due to derangement of other digestive organs. A very short review of the functions of the liver, pancreas, and intestines is thus imperative.

The Biliary Secretion.—Bile is both a secretion and an excretion of alkaline reaction. It is viscid from the presence of much mucus. It contains biliary acids and pigment as well as cholestrin, soaps, and fats. There is an enormous output in twenty-four hours—from 500-600 cubic centimetres being excreted. This is often demonstrated in the capacity that some subjects of gastro-enterostomy have of vomiting enormous quantities of bile. It completes the work of the pancreas in emulsifying fats, and it facilitates their absorption. If fatty acids be too abundantly formed, bile dissolves them. Constipation is prevented by its action in stimulating the peristalsis of the colon.

The acid chyme which comes into contact with the bile is reacted on by the latter secretion. The pepsin and, probably, the albuminoids are precipitated. These are rapidly re-dissolved and absorbed. In the meantime the reaction of the chyme is gradually changed from acid to neutral or faintly alkaline. Bile aids the pancreas in the emulsification of fats and, in cases of obstructive

jaundice, when bile is largely absent from the intestine, there is a great diminution in the utilization of fats. Although it is a disputed point, it is generally accepted that bile has specific antiseptic properties and prevents fermentation and putrefaction. This is, however, not wholly true, for it should be borne in mind that bile cannot be regarded as a universal germicide. It is indeed one of the best culture media for the enteric bacillus, and gall stones, originating through the action of organisms, form in the gall bladder.

The Pancreatic Secretion is alkaline, odourless, viscid, and highly albuminous. It owes its alkalinity to sodium carbonate and phosphate. It is the most important of the digestive secretions, and it contains three active ferments :—

(1) Trypsinogen. When this pro-ferment comes into contact with the duodenal secretion (Enterokinase) it is converted into the active ferment Trypsin. Enterokinase probably dissolves the proteid portion of the trypsinogen and thus sets free the trypsin. Clinical experience, however, shows that pancreatic secretion is a powerful digestive agent without coming into contact with and being activated by enterokinase. A troublesome surgical complication sometimes follows operation on this gland by the establishment of a fistula. The escaping pancreatic secretion practically digests everything with which it comes into contact—the skin and even the pancreatic tissue itself.

Trypsin is active in alkaline, neutral or slightly acid media. It digests quickly and completely the proteids and changes them into albumoses and peptones. From the latter polypeptids are formed, and these in turn are broken up into leucin, tyrosin, and tryptophan. Protein substances are ultimately analysed into amino-acids, in which form they are absorbed.

(2) Amylopsin or Amylase splits up starches into maltose and then into glucose, while cane sugar is converted into grape sugar.

(3) Steapsin or Lipase is a fat-splitting ferment of the pancreas. Fats are broken up into glycerin and fatty acid. The latter combines with the intestinal alkaline secretion and forms soluble soaps. These in their turn aid the emulsification and, therefore, the absorption of fats. These actions are also promoted by the influence of the bile.

It is important to note that the secretion of the pancreas and liver is promoted by the action of the secretions absorbed from the

duodenum and jejunum, and carried, *via* the circulation, to these organs.

If from a too rapid evacuation of the acid chyme into the duodenum the biliary and pancreatic secretions are overwhelmed and cannot sufficiently neutralise the stomach contents, the pylorus automatically closes, and thus more chyme is prevented from passing till that already in the duodenum is adequately neutralised. When this takes place the pylorus relaxes and permits the passage of a further supply from the stomach.

According to Barclay's observations, in his Radiographic Study of the Alimentary Tract, there would appear to be an intimate reflex connexion between the last part of the ileum and the pylorus. When the last part of the small intestine becomes distended with food, reflex closure of the pylorus occurs to prevent further overloading of the gut. When there is disease of the cæcum, this reflex is disordered and the automatic regulation of the intestinal supply becomes arrested. This very valuable observation affords an explanation of the dyspeptic symptoms so commonly accompanying appendicitis.

The Succus Entericus.— The chyme is now subjected to the action of the intestinal juice. This secretion contains water, mucus, albumin, and salts. It is alkaline in reaction, and exerts a neutralising influence on the acids formed by fermentation in the intestine — more particularly those resulting from the fermentation of starches. The succus entericus supplements the action of the trypsin of the pancreatic secretion, and the digestion of starches and fats is promoted. It contains certain special ferments :—

(1) *Enterokinase* appears to be a product formed by the action of other enzymes. It activates trypsinogen and perhaps other intestinal ferments.

Erepsin, of the intestinal juice, is a reserve ferment to complete the digestion of certain proteid products prepared by the previous actions of gastric and pancreatic digestion. It has little or no action on the proteins as they occur in our aliment. It acts on protein and it breaks up peptone into leucin and tyrosin.

There are also a number of sugar-splitting ferments :—

Invertase converts cane sugar into dextrose and fructose, or the disaccharids into monosaccharids.

Lactase converts milk sugar (lactose) into dextrose and galactose

Maltase converts the maltose, formed by the action of the ptyalin, into dextrose.

The action of the essential ferments in the intestinal tract is modified and enhanced by the influence of certain micro-organisms. These cause fermentations, and perhaps play an important rôle in destroying other organisms which might be harmful. Under certain conditions, when intestinal organisms, more or less normally present, become greatly augmented in number, pathological changes may result, and products, of the character of ptomaines, etc., be formed and absorbed, producing various degrees and forms of auto-intoxication. The subject of gastro-intestinal sepsis and auto-intoxication is one of much importance. It is quite possible, nay probable, that these intoxications have certain etiological relationships to cirrhosis of the liver and to certain renal affections, while collateral infections of other parts, by the bacillus coli, as for example the bladder, are of common occurrence.

The bacillus lactis aerogenes splits up the carbo-hydrates, producing ethylic alcohol and many acids—such as lactic and acetic. Formed in proper amounts, these acids have a benignant action, and check carbohydrate decomposition and stimulate peristalsis.

Aliment so prepared is readily absorbed. Fats enter by the villi, and find their way into the thoracic duct, which discharges into the venous circulation at the junction of the left subclavian and jugular veins. The other substances are absorbed through the capillaries in the villi, and hence into the general circulation *via* the portal vein. The chyme is rapidly passed down the tract and, in normal conditions, has traversed the entire small bowel in the short period of from two to six hours after the ingestion of food into the stomach. By far the largest amount of absorption takes place in the small intestine, and only a small amount of soluble nutriment remains when the chyme reaches the large bowel. As far as the splenic flexure it is fluid. Below the splenic flexure it rapidly becomes drier, till it arrives at the rectum, where it forms a semi-solid or pultaceous mass of fæces.

Normally, one evacuation of waste material should take place daily from the bowel. It is composed of insoluble detritus such as cellulose, bacteria and epithelial cells. The average adult stool weighs about five to six ounces. It contains from 65 to 75 per cent. of water and from a quarter to one-third of its weight, when dried, is due to bacteria.

Its passage down the colon is facilitated by the abundant mucus secreted by this part of the gut. Except for the action of micro-organisms on cellulose, no further digestive process is contributed by the colon.

Hyper, or diminished, intestinal motility gives rise to many disorders—such as diarrhœa, constipation, and auto-intoxication. The character and amount of the chyme in the intestine are intimately related to the intestinal motility. Thus it is that diet is often at fault in certain cases of constipation or diarrhœa.

Water is absorbed by the to-and-fro movement of the chyme in the ascending colon. But although absorption of water with salts and any soluble albumen does take place from the ascending and perhaps transverse parts of the colon, the descending part and rectum possess little absorptive power for nutriments. The lower part of the colon can take up water and salts with great rapidity, especially if the circulation be depleted and the tissues to any extent dehydrated, but it is questionable if it can absorb nutrients in their normal condition. From numerous observations I am convinced that complex nutrient enemata are worse than useless. They undergo rapid putrefactive changes in the rectum, and by-products are absorbed which produce intoxication, resulting, for example, in acetonæmia. Normal saline solutions and glucose are readily absorbed, and the benefit which has been stated to accrue from nutrient enemata is probably entirely due to the water and salts they contain.

A careful study of the fæces is very essential for the elucidation of many gastro-intestinal derangements. Because, perhaps, a careful macroscopic and microscopic inspection offends our æsthetic taste, such an examination is far too often omitted, and many avoidable errors in diagnosis are thus made. A detailed account of the clinical examination of the fæces will be given later on.

CHAPTER III.

ANAMNESIS.

Examination of the Patient.

Till comparatively recently no attempt was made to make a scientific examination of the patient suffering from symptoms of gastric derangement.

No progress in diagnosis or treatment can reasonably be expected till all methods of examination are brought to bear on a given case. So many symptoms are common to several different ailments that in every case the necessity for a complete examination is very apparent if a correct diagnosis is to be obtained.

Because a patient describes symptoms indicative of digestive derangement, the physician will not conclude, till after thorough investigation, that these are primarily due to a fault in the gastro-intestinal organs.

Many general and special diseases of other organs than those of the digestive system may produce stomachic symptoms. Chronic bronchitis and emphysema, many valvular diseases of the heart, disorders of the central nervous system, of the kidneys, ovaries, and uterus are often associated with urgent symptoms of gastric disorder.

Most people who suffer from any severe chronic affection of the stomach acquire the physique of malnutrition. The skin is apt to become 'muddy,' the nose is frequently the seat of Rosacea, and many skin irritations may make their appearance. Thickening of the second phalangeal joints is, according to Bouchard, almost diagnostic of chronic gastro-intestinal auto-intoxication.

At the bedside it is well to have a definite scheme of interrogation and investigation to aid the elucidation of these conditions. As far as possible the patient's own story should be patiently listened to, but, as very accurate details are required for diagnosis, certain questions must be asked.

I have found the following scheme useful for keeping records of gastro-intestinal affections :-

Gastro-Intestinal History Record.

I. **Name of Patient,** age, occupation and address.

II. **Date of Examination and History.**

III. **Family History.**

IV. **History of Past Illnesses.**

V. **Habits and Social Condition.**
- (1) Time allowed for food ?
- (2) Is food eaten at work ?
- (3) Nature of diet ?
- (4) Tea, coffee, tobacco or alcoholic habit ?
- (5) Hygienic surroundings ?

VI. **History of Present Illness.**
- (1) Exact Symptoms complained of. in patient's own words ?
- (2) Exact Mode of Onset ?
- (3) Appetite ?
  - Normal ?
  - Diminished ?
  - Anorexia ?
  - Increased ?
  - Perverted ?
- (4) Nausea ?
  - Relationship to food ?
- (5) Vomiting ?
  - Time of onset ?
  - Frequency ?
  - Severity ?
  - Are symptoms relieved by act ?
  - Accompanied by nausea ?
- (6) Characters of Vomited Material ?
  - Colour ?
  - Quantity ?
  - Taste ?
  - Odour ?
  - Frothy ?
  - Mucus ?
  - Blood ?
  - Any other distinctive feature ?
- (7) Pain ?
  - Positions of ?
  - Time of onset ?
  - Duration of ?
  - Character of ?
  - Is it relieved or aggravated by food, fluids, or medicines ?
  - Effect of pressure on ?
  - Effect of posture on ?
- (8) Flatulence ?
  - Time of occurrence ?
  - Belching or Borborigmi ?
  - Associated with vomiting or pain ?
- (9) Heartburn ?
  - Time of occurrence ?
  - Influence of diet on ?

(10) Eructations of Stomach Contents ?
Time of onset ?
Taste of ?
(11) Vertigo or Faintness ?
Time of onset and degree of ?
(12) Emaciation ?
Time of onset ?
Progress of ?
Degree of ?
(13) Anæmia ?
Character and degree of ?
(14) Headache ?
Situation of ?
Character of ?
Time of onset of ?
(15) Condition of Bowels ?
Size of Stool ?
Number ?
Odour ?
Colour ?
Shape ?
Recognisable fat ?
Diarrhœa ?
Constipation ?
Mucus ?
Blood ?
Worms ?
Shreds ?
Recognisable food particles ?
Pain on defæcation ?
(16) Deglutition ?
Normal ?
Abnormal (Dysphagia) ?
(17) Cardio-Vascular Disturbance ?
Palpitation ?
Facial flushing after meals ?

VII. **General Examination.**
Weight ?
Condition of Teeth ?
Condition of Tongue ?
Condition of Naso-pharynx ?
Abnormalities in other systems?

VIII. **Physical Examination of Abdomen** (during fasting and after food).
(1) Inspection ?
(2) Palpation ?
General ?
Local ?
Examination of Rectum ?
(3) Percussion ?
General.
Local.
Auscultatory ?
(4) Auscultation ?
Friction over liver, spleen, etc. ?
(5) Auscultatory Percussion ?
(6) Mensuration ?
(7) Inflation of Stomach and Colon ?
(8) Radiography ?

IX. **Investigation of Gastric Motor Functions.**
Tests employed ?

X. Chemical Examination of Stomach Contents, Fæces, and Urine.

(1) Stomach Contents (after test breakfast or dinner).

- Reaction ?
- Free Hydrochloric Acid ?
- Combined Hydrochloric Acid ?
- Total Acidity ?
- Lactic Acid ?
- Other Organic Acids ?
- Occult Blood ?
- Pepsin ?
- Rennet ?

(2) Fæces.

- Reaction ?
- Fat (Free and Emulsified) ?
- Pancreatic Efficiency ?
- Bile ?
- Blood—Macroscopic or Occult ?
- Mucus ?

(3) Urine.

- All the ordinary tests.
- Indican ?
- Acetone and Diacetic Acid ?
- Chlorides ?

XI. Microscopic Examination of Gastric Contents, Fæces, etc.

(1) Gastric Contents.

- Starch ?
- Meat Fibres ?
- Mucus ?
- Pus ?
- Infusoria ?
- Cancer and other Cells ?
- Yeast (discrete and in chains) ?
- Boas-Oppler Bacilli ?
- Sarcinæ Ventriculi ?
- Lactic Acid Bacilli ?
- Tumour Tissue ?

(2) Fæces (after tritration with distilled water, if solid, drop on slide with cover-slip. Examine without and with Iodine staining).

- Fat (with Glacial Acetic Acid and heat for Fatty Acid Crystals ?
- Undigested starch ?
- Pus ?
- Blood ?
- Mucus?
- Meat Fibres ?
- Oxalate of Lime ?
- Tumour Tissue ?
- Organisms ?
- Parasites ?

XII. Diagnosis.

XIII. Treatment.

XIV. Progress and Result.

In connexion with the above scheme for taking the history record of a gastro-intestinal case, too much patience and care cannot be employed to obtain correct answers to the various questions.

Much light will thereby be thrown on the nature of the disease and a diagnosis be facilitated.

It is only intended here to illustrate the importance of a careful clinical history and the elucidation of the points in the history record without going into all the considerations which this investigation may open. In discussing the diseases and disorders of the stomach these will be dealt with more in detail, and their import indicated. The aim of the scheme of the gastro-intestinal history record is to obtain data on which a diagnosis may be based.

Age.—It need hardly be said that in childhood most gastric troubles are due to errors in diet. They are apt to be acute and associated with toxic symptoms often of a severe character. Young women, especially if anæmic, are liable to acute gastric ulceration. Duodenal and chronic gastric ulcers are commoner at a more advanced age—*e.g.*, from 20 to 40. Carcinoma, as a rule, occurs later in life, but from time to time it is found in the comparatively young.

Occupation has often a close relationship to gastro-intestinal affections. A sedentary life and the stooping posture promote constipation and many digestive derangements. Gastric ulcers are very common among machinists, tailoresses and dressmakers, who have little healthful exercise, who too often carry their food and work in insanitary surroundings.

Sex.—Gastric ulcers are commoner in young women than in men because, among other causes, they are more liable to anæmia. Various gastric neuroses have also a sex incidence, and many of them, such as Achylia Gastrica Nervosa, occur more frequently in women than in men. Duodenal ulcers and gastric carcinoma are, however, commoner in men than in women.

Social Habits and Conditions.—Generally these have an important etiological bearing in diseases of the gastro-intestinal tract. The evil of too little time allowed for the meal-hour, resulting in improper mastication, needs hardly any comment. Certain derangements arise from the habit of eating food while at work. Many industrial diseases may thus be produced—*e.g.*, lead poisoning. Meals that are carried are probably never so digestible as freshly prepared food. The nature of the dietary should be carefully ascertained in investigating any case. When improperly prepared and unsuitable food is taken for long periods of time, sooner or later, digestive

disturbance will ensue. Among the better classes there is a tendency to eat too much and too many things at one time. Too great strain is thrown on the gastro-intestinal digestive capacity, and any dietary which is in excess of the requirements of the individual overtaxes the excretory organs and waste products are improperly removed. Their presence in the blood is responsible, not infrequently, for a variety of constitutional diseases such as gout, renal affections, and arterio-sclerosis.

Equally important is a consideration of the nature and amount of stimulants and narcotics habitually taken. Strong Indian tea, taken almost at every meal, as it is among the working classes, can be nothing else but injurious. Each cupful is a dose of tannic acid possessed of strong astringent properties, which tends to produce constipation. Harmful, when too strong and in too large quantity, at any time, it is specially so when taken with fresh meat. Tannates of albumen, when formed in moderate amount, are capable of resolution and absorption; but when in excessive amount the nutrient value of the meal is considerably impaired. The constipation and defective nutritive properties of the food are fruitful sources of anæmia and gastric ulceration and of other derangements of the stomach.

The abuse of alcohol and tobacco may produce not less serious results. Alcoholic gastritis, ending in atrophic changes of the stomach mucosa, is commonly met with as a cause of permanent achylia.

The social conditions in which many patients live are also frequently conducive to various diseases. Bad hygienic surroundings tend to produce anæmia and digestive troubles.

Family History.—The more experience one has of gastric affections, the more convincing is the evidence that these disorders are frequently familial. From my case records I find that it is no uncommon occurrence for several members of one family to have suffered from gastric or duodenal ulceration or from dilatation of the stomach. This has been specially true of the male members of these families. The history of previous general or digestive disorders may be informing. Many digestive derangements are recurrent and relapsing—such as dyspepsia of phthisis, gall-stones, and peptic ulcer. Others are associated with a record of past good health and a perfect digestion, as in cancer of the stomach.

The History of the Present Illness cannot be too carefully gone into. As far as possible a statement of the patient's complaints should be got in his or her own words without any leading questions being put. It is imperative in all cases to ascertain if any symptoms referable to the digestive tract pre-existed, or if the present attack is rather only of the nature of a relapse.

The exact mode of onset is often very suggestive. In gastric ulceration a history of chronic digestive derangements will commonly be offered or ascertained by cross-questioning, while in carcinoma of the stomach a history of recent disturbance, in a patient with a previous good digestive record, must always be regarded with suspicion as suggestive of malignant disease.

The *appetite*, in carcinoma is almost invariably defective or lost, but in simple ulceration it is commonly maintained, or may even be increased. In many *neuroses* the appetite becomes perverted and capricious.

Nausea is a frequent symptom of a great variety of functional and organic digestive diseases. Its time of onset and its relationship to the ingestion of food will usually afford valuable information as to its causation. It is commonly found in most cases of carcinoma, and is often very persistent; but not infrequently this symptom may be entirely absent in gastric and duodenal ulcerations.

Vomiting is even more important than nausea, and when it occurs a number of points require consideration. Its relationship to food is of great importance. In most gastric troubles it occurs with a definite periodicity corresponding to a meal. In gastric ulceration, when it occurs, it comes on very soon after the meal has been taken, and is unusually delayed beyond two hours. In duodenal ulceration it is greatly delayed, and corresponds to the act of the passage of chyme through the pylorus.

Pain.—Pain is a most important symptom. Care must be taken to determine its character and estimate its significance.

The situation of the pain.—Generally speaking pain above the umbilicus or in the gastric region may be referred to the stomach, while pain below the umbilicus has usually an intestinal origin. The exact location is often significant. In ulcer inside the stomach the pain is commonly localised to the left epigastric region, while in ulcer at the outlet it is situated to the right of the middle line in this region. It should be recalled that pain is often referred or reflex, and besides starting at the seat of the lesion it may have

a wide distribution in a well-defined manner. Such, for example, are the pains complained of in the spinal and scapular regions in gastric and hepatic disorders.

The *time of onset* of the pain not infrequently gives valuable information of the situation of the lesion. In ulceration at the outlet the pain is delayed till the act of digestion is nearing completion, and the stomach beginning to empty. In peptic ulcer, well inside the viscus, the pain may quickly follow ingestion; but in duodenal ulcer two to four hours may elapse before pain is experienced.

The *duration of pain* should also be noted. Does it only persist during the act of digestion, or is it more or less constant? In tumour formation the pain may be persistent, and not be related to ingestion at all.

All degrees of *severity of pain* may be complained of. When pain is very persistent in peptic ulcer it may be inferred that the peritoneal layer is implicated and that perforation may not be far off. In my experience pain of a severe and persistent character is present in duodenal ulcers which have implicated the pancreas. Such cases require very careful watching.

All the *circumstances which modify the pain* or relieve it should have consideration. What is the exact influence of ingestion on the pain? In duodenal ulcer a meal often temporarily gives relief till the stomach begins to empty itself.

The so-called *hunger pain*, which certain authors regard as almost diagnostic of duodenal ulcer, occurs shortly before the next meal is due, or even more frequently during night, between 12 and 4 a.m., when the stomach is most empty. While it is my experience that hunger-pain is a very common symptom of duodenal ulcer, too much reliance should not be placed on it alone. I have found that it frequently may accompany marked hyperchlorhydria or gastrosucchorrœa when no erosion exists.

Normally gastric secretion ceases during fasting, but in duodenal ulcer the constant irritation accompanying the lesion induces a perpetual supersecretion, even when the stomach is quite empty.

Hyperchlorhydria of nervous origin may be accompanied by hunger-pain. In short, if the secretory activity of the stomach persists during fasting, a gnawing discomfort or pain, with craving for food, is experienced in the gastric region—the so-called *hunger-pain*. When food is introduced the over-abundant active secretion

is utilised or ' combined ' and the irritation which it causes to the empty stomach forthwith ceases.

In peptic ulcer inside the viscus, pain often is immediately induced by eating, and is maintained till the stomach is emptied either naturally or by vomiting. A drink of fluid—milk, water, etc.—will not infrequently temporarily bring relief in peptic ulcer, especially in ulcers at the outlet. Patients soon get to know that alkalies relieve the pain, and a draught of hot water in which a teaspoonful of sodium bicarbonate is dissolved is a popular, though harmful, remedy. The alleviation of pain following the ingestion of food or alkalies is due to the utilisation or neutralisation of the over-acidity commonly accompanying peptic ulcer, as well as to the arrest of peristaltic movements and over-tonicity which accompany hyperchlorhydria. In malignant disease of the stomach the conditions are different, and the pain is less influenced by food and drugs.

The *posture* of the patient may also influence or modify gastric pain. In the pain due to adhesions and peptic ulcer this is often the case. When adhesions exist they are commonly dragged upon in the erect position. The pain of ulcer may be modified by altering the posture. In ulcers at the outlet the pain may be relieved by getting the patient to lie on the left side, while in ulcers at the cardia it may be modified when the patient lies on the right side. When the ulcer is located on the posterior wall of the smaller curvature—its most usual situation—pain is relieved on the assumption of the erect posture.

In cancer of the stomach *pressure* (as lying on the belly over a pillow) rather aggravates the pain, while in peptic ulcer it may relieve it.

Flatulence.—Flatulence is a most pronounced symptom in gastric stasis with fermentation. It is thus usually a prominent feature of chronic simple and malignant stenosis.

In hysterical and highly neurotic subjects air may be swallowed in large quantities, and as constantly be noisily belched. Such cases, as a rule, are unassociated with organic lesion, and are easy of recognition.

Flatulence occurs also in achylias and in many cases of hyperchlorhydria with amyldyspepsia. The odour of the gas eructated is sometimes characteristic. Sulphuretted hydrogen is occasionally eructated in simple stenosis, and an offensive gas of putrefaction in advanced cancer.

The patient who complains of flatulence should be questioned as to whether it is belched up or is passed by the bowel, and whether evacuation of wind brings relief. The time of its occurrence should also be noted.

An enquiry into the presence or absence of symptoms of Acidity must also be undertaken. It is not always present even when the patient asserts that it is his trouble. It may merely be his interpretation of his symptoms. As a rule pronounced heartburn is easily recognised. A history of its temporary relief on taking solid or liquid food or alkalies is almost diagnostic. But the practitioner should not assume that heartburn is always due to hyper-chlorhydria. It is a usual accompaniment of that abnormality, but it often occurs in a more inveterate form in malignant achylia, from organic acids, where the *absence* of free hydrochloric acid permits of the formation of these.

Not uncommonly fluid eructations of the stomach contents accompany the belching of wind. The nature of the disorder may be suggested by taking the reaction of these with blue litmus and congo-red papers. If the litmus is sharply reddened and the congo-red shows no bluing, the sour eructations are due to organic acid. If the blue litmus be reddened and the red congo turned blue, the sour mouthfuls contain hydrochloric acid.

Vertigo and faintness and many vaso-motor disturbances may result from flatulent distension of the stomach as well as from toxæmia from the absorption of abnormal ingredients.

Emaciation has to be considered with reference to its time of onset, its progress and degree. In all severe, even temporary, gastric disorders with loss of appetite and failure of the digestive functions, weight is sure to be lost, and too much importance must not be placed on this sign. But when loss of weight has a very definite onset, is progressive and great, it is always an index of a serious malnutrition, and, if other diseases associated with emaciation can be excluded, it favours an organic lesion of the stomach. It is most pronounced in malignant disease, especially at the pylorus, and is present in all cases of serious retention of the gastric contents.

Anæmia.—There may be a minor anæmia as part of the constitutional disturbance due to the general malnutrition of any gastro-intestinal disorder. The continuous absorption of toxic substances from the digestive tract certainly has a deteriorating

influence on the blood. But apart from these minor anæmias we may have the bloodlessness becoming a prominent symptom, and producing in itself disturbances of a grave character.

The nature, degree, and mode of onset of the anæmia may require careful investigation. If pronounced anæmia occurs suddenly, it usually indicates a profuse hæmorrhage, and will be associated with melæna and perhaps hæmatemesis. Profound chronic progressive anæmia of slow and indefinite onset is characteristic of cancer of the stomach. It will be prudent in all the latter types of anæmia to make a blood examination to exclude the pernicious variety from that of malignant disease of the stomach.

Headache.—All gastro-intestinal disorders may be associated with headache, most commonly due to auto-intoxication. The pain is usually frontal.

Condition of the Bowels.—In obtaining the history of a gastro-intestinal disorder this important topic must be gone into with care. Many people will assert that their bowels are moved with regularity when perhaps there is only evacuation twice weekly or even less frequently. Such a form of " regularity " is of course not normal, and is likely to give rise to various disturbances.

A statement giving exact facts must be obtained, if possible. So many disturbances attributed to a disorder of the stomach are referable to constipation that before diagnosing a gastric disorder we should exclude this condition as a possible cause. Some people are habitually constipated, while others suffer from looseness of the bowels or diarrhœa without apparent disturbance of the health. Usually, pronounced diarrhœa is abnormal, and is associated with abdominal disturbance or general debility. The reader is referred to the chapters on constipation and diarrhœa for a fuller consideration of these subjects.

Much has recently been said and written of the widespread evils of intestinal stasis, and although there is a tendency to attribute too far-reaching pathogenic influences to this cause, it undoubtedly is responsible for many of the common ailments of life.

While one evacuation in twenty-four hours may be regarded as normal, it not infrequently happens that certain healthy subjects have two motions daily, and it is not very uncommon to find that some individuals, apparently in excellent health, have the bowel relieved only twice weekly, or even at much longer intervals, without any signs or symptoms of auto-intoxication.

The more pronounced the constipation the greater the tendency for the motion to be formed of scybalous masses.

In different individuals the size of the motion varies. Of course there is a most direct relationship between the amount of fæces evacuated and the amount and kind of food ingested. The rougher the diet, and the greater the amount of indigestible and insoluble material it contains, the larger the detritus left.

Very bulky stools, apart from those produced by a rough diet, are often pathologic. In advanced pancreatitis, with failure of pancreatic secretion, intestinal digestion is in abeyance and the aliments are inadequately dissolved and absorbed. A very large stool, out of all proportion to the amount of food ingested, results. Such a stool will have other abnormal characters, which are detailed under Pancreatitis (see page 339) as, for example, the presence of recognisable fat.

The normal round shape of the stool is determined by the lumen of the colon and partly by moulding as the fæcal mass passes through the sphincter. If the fæces be soft and pultaceous in character the motion will be small in diameter, and, if there be any obstruction such as piles at the outlet, the motion may be ribbon-shaped or otherwise distorted. The latter features, unless persistent, have not to be regarded as evidence of obstruction.

The colour of the fæces in healthy adults, on a mixed diet, is ordinarily light brown. But very many conditions apart from actual disease may contribute to alter the colour of the stool. The longer a motion lies in the colon the more dark and dry it is apt to become.

Moreover the nature of the diet greatly influences the colour of the fæces. From the light canary or mustard colour of the stools of infancy, and of adults on a diet confined to milk, we have all gradations in tint to that of a dark brown. The larger the amount of meat and nitrogenous foods taken the darker will the stools be. Indeed, if meat be taken in excess, the stools may almost approach a black colour.

Charcoal produces a very characteristic intense black motion. It may sometimes be used to ascertain the rate at which the chyme passes through the digestive tract.

Bismuth tints the stool a blue-black colour quite easy of recognition.

Blood, when fairly abundant, produces a tar-coloured stool,

and if very copiously present the consistency, as well as the colour, resembles tar. The blackness of the stool (melæna) is due to blood altered by the action of the digestive fluids, and to the sulphuretted hydrogen acting on the hæmoglobin and forming black sulphide of iron. The student must not fall into the error of supposing that the absence of melæna constitutes the absence of blood. Even the palest stools may contain an appreciable quantity which can easily be detected by chemical analysis (occult blood).

Frankly red stools with unaltered blood, usually mean that the bleeding spot is low down in the tract, and that the blood has been evacuated before there has been time for alteration in its colour.

It should always be carefully noted whether the red colour, due to blood, is intimately mixed with the stool, as it may be when the latter is liquid, or whether it merely coats the surface, as it does from hæmorrhage from piles and fissures, during the passage of the motion.

The odour of the motion is described as " fæcal." But it varies greatly in health in different individuals, and, from time to time, in the same individual from modifications in diet and health. The normal smell is principally due to skatol, indol, and various gases. The healthy evacuation should not be otherwise offensive. Very foul-smelling stools usually indicate putrefaction from certain abnormal conditions.

The stools in pancreatitis and in obstructive jaundice may be very offensive. Many morbid conditions, such as enteric fever and various constitutional diseases, as pernicious anæmia, are associated with the evacuation of foul-smelling stools.

A sour smell usually indicates the presence of excessive quantities of fatty acid. This is commonly met with in infancy from decomposition of the milk, and in adults who are suffering from carbohydrate fermentation.

When nitrogenous putrefaction is prominent, the stools are very fœtid in smell.

Mucus is not present in quantity in the normal stool passed spontaneously ; but it is often apparently excessive in a motion produced by a large enema. In the Plombière treatment the lavage of the colon with very large volumes of water causes the evacuation of various amounts of an unorganised mucus which should not necessarily be—although it often is—regarded as pathologic and

indicative of colitis. *Abundant mucus passed spontaneously and habitually* with the stool is pathologic and indicates a catarrhal condition of the colon. The exact character of the mucus should be noted, as well as its relationship to the fæces. Small amounts of mucus coating only the surface of the motion are not significant of disease. Sometimes when abundant and pathologic the mucus may be evacuated apart from the stool, or, if accompanying it, may not be incorporated with it.

Mucus may be glary (unorganised) and starch-like, or it may be stained yellow from the fæces, or red from admixture with blood.

In dysentery and post-dysenteric colitis large quantities of jelly-like mucus, stained with varying amounts of blood, may be passed ; or mucus may be partially desiccated or coagulated and form a mould or cast of the bowel (membranous colitis).

The reader should be on his guard in making a not uncommon mistake in diagnosing ' mucus colitis ' when a more grave condition may be present. In people, men particularly, at or about the age of fifty, who have been in good health previously, the habitual passage of any considerable quantity of mucus or blood-stained mucus should not be regarded as mucus colitis till malignant disease of the rectum or colon has been excluded. This grave condition should always be considered and a rectal examination with or without the sigmoidoscope invariably made.

The help of X-rays and an opaque meal or enema may be also invoked in cases of doubt.

Parasites, as thread, round, and tape worm, may be passed per rectum. These are usually recognised if a little care be taken, but before their existence is diagnosed they should have been seen by the physician. Patients often state they have passed worms when these parasites are not present. Shreds and certain fibres may, to the inexperienced, resemble one or other of the worms.

A normal stool ought to be fairly homogeneous, and when the chyme has been well digested recognisable particles of food should not be present. In infants suffering from gastro-intestinal derangement the fæces often contain unaltered milk curd.

Sometimes Gall-stones of various sizes and shapes may be obtained from examination of the fæces. After an attack which may be regarded as biliary colic it is most essential to examine the

stools for three or four days after the seizure. The fæces, if solid, should be broken down in water and sieved through muslin over a water-closet. All the insoluble debris should be kept for the physician's inspection.

A complete investigation into the condition of the bowels will include an enquiry as to the *presence or absence of pain before, during, or immediately after defæcation.* Colicky pain before evacuation may be due to rectal or colon obstruction from growth, ulcer, or spasm. Pain during the act is commonly due to piles, fissures, or cancer, close to the orifice. Sometimes it may arise from enlargement or inflammation of the prostate gland. Pain after defæcation may have its origin in any or all of these conditions. When pain is persistently an accompaniment of defæcation, a thorough rectal examination is always demanded.

Attention should always be given to the act of deglutition. Any pain or difficulty in the swallowing will be noted, and subsequent examination, to determine its cause, will be required.

Before closing the investigation of the symptoms, enquiry should also be made as to the *effect of a gastro-intestinal derangement on the cardiovascular action.* Mechanically, from over-distension of the stomach by gas, the heart's action may be disturbed. Violent palpitation is not an infrequent accompaniment of flatulent dyspepsia. The heart is greatly embarrassed in the meteorism of enteric fever when abdominal distension is great. Arhythmia and a tendency to syncope are apt to accompany this condition. If cardiac disease be excluded, a complaint of pre-cordial distress (pseudo-angina), with palpitation and dyspnœa, may frequently be assigned to a gastric disorder.

Facial flushing and other vaso-motor disturbances are not uncommonly associated with digestive troubles. The Rosaceas and other vaso-motor skin rashes accompanying dyspepsia have their origin thus.

Skin affections are also due to auto-intoxications. Acne is commonly met with in all cases where fats are badly digested and fatty acid is formed in excess.

Oral Cavity.—In considering any form of digestive derangement a careful examination of the buccal cavity and naso-pharynx should never be omitted. A careless survey often fails to detect lesions whose presence might throw light on certain morbid symptoms.

The mucous membrane of the buccal cavity ought generally to be scrutinised to ascertain its colour and the presence of any abnormality. An anæmia, not remarked by examination of the skin, may be detected by undue pallor of the mucous membrane of the mouth.

Cyanosis can here be readily seen. There is no better way of detecting minor and doubtful icterus than by compressing the lip under a microscopic slide till it is completely blanched and observing, in daylight, if any yellow tint be present.

(1) The odour of the breath is often informing. When Pyorrhœa Alveolaris is present the breath is constantly and characteristically fœtid. If this condition be suspected, a piece of paper, or floss silk, passed between the gum and the neck of the tooth will, on withdrawal, be saturated with the disgusting odour of this condition. Ozœna and purulent inflammation of the throat also make the breath foul. If the breath is fœtid only some few hours after ingestion, it is probably due to a digestive derangement, and is often associated with putrefaction of the food.

It is habitually heavy in chronic constipation.

Certain general disorders of the system are associated with foul breath. Most of the eruptive specific fevers give rise to a fœtid breath. In young children suffering from gastro-enteritic disturbances, acetonæmia with characteristic breath is frequently present, and the presence of diabetes can sometimes be suspected from the sweet odour of the breath. In uræmia, alcoholic and opium poisoning, the breath is frequently diagnostic. It is very foul in mercurialism, and very fœtid in pulmonary gangrene.

(2) A bad taste in the mouth is often associated with a foul breath, and is most common in the morning. Abnormal tastes for certain things occur in hysteria, and in chronic gastro-succorrhœa there may be a distaste for certain things formerly liked.

(3) The lips and gums, in carcinoma of the stomach, and in other diseases associated with severe auto-intoxication, are apt to become covered with sordes, and get dry and hacked.

(4) The importance of sound teeth and a clean mouth has already been commented on. Many diseases take origin in unhealthy conditions of the mouth.

The teeth form a very important part of the digestive apparatus. Their condition demands careful scrutiny. Bad, or an insufficient

number of, teeth sooner or later may produce serious digestive derangement as well as other diseases.

Besides the characteristic malformations due to rickets (rachitic teeth) and syphilis (Hutchinson's teeth), the presence of caries and pyorrhœa alveolaris ought to be sought for. There is little doubt that oral sepsis may play a pathogenic role in certain of the common organic lesions of the stomach and duodenum.

The gums may become inflamed, bleeding, and spongy from overdoses of certain drugs—*e.g.*, potassium iodide and mercurial preparations. Here, too, the presence of lead in the system may be detected by the blue line in the gum at the neck of the teeth. This is due to a deposit, in minute particles, of the black sulphide of lead. In people who neglect to care for their mouths, and in heavy smokers, the teeth are often discoloured at the cervical region, and a pseudo-blue line is thus formed. It is quite easy to differentiate the true from the false by inserting the edge of a thin calling-card between the tooth and the gum and observing whether the blue line be *in*, or merely the black discoloration of the teeth shining through, the gum. In the latter case the line disappears at once from the portion of the gum separated from the teeth.

(5) The tongue is not always an infallible index of the condition of the digestive organs. Some apparently healthy individuals have habitually dirty tongues. Alterations in the colour and coating of the dorsum, produced by medicines or smoking, should be remembered.

The tongue is, however, usually a faithful reflector of certain gastro-intestinal and constitutional diseases. In some instances, it is true, it may remain clean even when these are present.

The movements of the tongue and the manner of its projection as well as alterations in its sensory condition call for note in certain lesions.

The amount of moisture or dryness is also of importance. A dry tongue is found in most febrile diseases, diabetes, and acute gastritis —especially in acute alcoholic gastritis.

More important in relation to digestive troubles is the coating which may be present on the tongue. The character of the fur or coat is sometimes informing. In chronic gastritis the dorsum is usually coated with a heavy yellow or greyish-yellow fur. In ulcer the tongue is often dry and red, and has a central whitish line. At other times the tongue is fiery red and smooth, as in the

early stage of acute gastrosuccorrhœa. In chronic hypersecretion the tongue is often red, moist, and very clean. It should, however, be remembered that the character of the tongue alone cannot be diagnostic of any disease of the digestive tract. The strawberry tongue of scarlet and the dry parrot-like tongue of enteric fevers are not wholly confined to these conditions.

The soft flabby tongue, indented round its margin by the teeth, coated thickly posteriorly, is suggestive of chronic gastritis. This character of the tongue is not unusually seen in the dyspepsia of chronic phthisis and in myasthenia gastrica.

CHAPTER IV.

DISEASES OF THE ORAL CAVITY.

The oral cavity is not infrequently the seat of various inflammatory diseases—chiefly of an infective character. These conditions are spoken of as stomatitis. They are most commonly met with in children, especially during dentition, but adults may also be attacked. They also occur with digestive and general disorders. The following types are well recognised.

(1) *Parasitic Stomatitis* (Thrush) is dependent on a fungus, Saccharomyces or Oidium Albicans. The disease is most commonly found in bottle-fed babies. It is characterised by the appearance on the tongue and buccal mucous membrane of pearl-white spots. These patches are at first discrete, but they ultimately may coalesce and form a uniform membranous-like growth over the entire buccal cavity. Later the fungus may extend to the gullet and descend into the stomach. While specially liable to attack infants and children, it is not uncommon to find the disease in adult life in connexion with chronic organic diseases of the stomach, or with grave constitutional disturbances.

(2) *Aphthous Stomatitis* (Maculo-Fibrinous Stomatitis) presents small round raised vesicles on a reddened base. These may be situated on the tongue or under the lips or on the mucous membrane of the cheeks. They burst and leave greyish-white ulcers surrounded by red areola. They are very painful, and often cause much salivation.

Aphthous Stomatitis may occur during any dyspeptic attack, catarrhal cold, or other debilitating constitutional disturbance. The condition is frequently recurrent during the course of grave organic gastric disease. In childhood, during the lactation period, it is most commonly caused by lack of cleanliness in the care of the feeding-bottles.

(3) *Ulcertive Stomatitis* is more commonly found in local and general infective conditions than associated with digestive derangements. It may be epidemic in institutions where the general

hygiene is bad. No definite pathogenic organisms have been isolated in this affection. The disease is not found in children prior to dentition. It appears to be due to the action, in most instances, of the Bacillus fusiformis, although a mixed infection may be present. The same organisms may be found in Vincent's Angina and in Noma, two conditions which, if not identical, must be closely allied to ulcerative stomatitis.

The lesion begins in the gum round the necks of the teeth. There is swelling and redness, and later ulceration with foul-smelling discharge. The mucous membrane of the cheeks in contact with the gums becomes implicated. There is usually loss of appetite and some constitutional disturbance.

(4) *Gangrenous Stomatitis* (Cancrum Oris, Noma) is not a feature of digestive derangements, and need not therefore be described here.

The Pharynx.—The pharynx is funnel-shaped, about four inches long in the adult, and widest transversely. It is composed of muscular and mucous coats—the latter being very rich in glands. The mucous membrane is continuous with that of the posterior nares and eustachian tubes. Above the nares the epithelium is columnar and ciliated, but below it changes suddenly into the squamous variety, which lines the remainder of the cavity as well as the gullet.

The various affections of the pharynx are not specially related to gastric diseases. But the pharynx should be carefully inspected to exclude possible gastric infections from this source.

Pharyngitis may be acute or chronic. The chronic disease may be the outcome of the acute, and both are frequently associated with inflammatory changes in the naso-pharynx. In so far as they are sometimes caused by digestive derangements, the functions of the stomach and bowels should be investigated and any abnormal condition treated.

Glossitis occurs at times as part of a catarrhal stomatitis. The tongue becomes swelled, painful, and its movements impaired, making articulation somewhat thick and deglutition difficult. There is a good deal of salivation.

Treatment of Oral Affections.—*Foul Tongue.*—If the tongue, during the course of a digestive trouble, be habitually thickly coated, the taste perception is impaired and appetite correspondingly diminished. It is sound practice systematically to clean the tongue

with a swab of gauze, soaked in lemon juice, and when as much as possible of the fur has been removed the mouth may be washed out with a little warm water to which has been added some glycerin of borax.

Fœtid Breath.—So many abnormal conditions may be responsible for bad breath that a careful examination of the entire buccal cavity and naso-pharynx must be undertaken. Any treatment adopted will depend on the cause of the fœtor. If due merely to want of cleanliness, the trouble may be removed by systematic use of the toothbrush morning and evening, and by the employment occasionally during the day of an antiseptic mouth-wash.

Too much attention cannot be paid to the preservation of the teeth by oral asepsis. It is unfortunately too true that the practitioner is often at fault in not insisting on the importance of the continuous care of the teeth from their very first appearance in the mouth. Not only does a foul breath result from want of cleanliness, but oral sepsis arises, which causes early decay of the teeth and lays the foundation for many functional and organic diseases of the stomach. A frequent visit to the dentist is of prime importance for the early detection of caries of the teeth.

Other morbid conditions must be sought out and appropriately treated. If the tonsils are diseased, and are the seats of cheesy deposits, they should be excised, bad teeth should be removed, and the nasao-pharynx treated if necessary.

Constitutional affections producing fœtid breath must be dealt with similarly. Constipation removed by laxatives, disorders of the stomach by dietetic and medicinal treatment, while pulmonary affections will be treated according to the disease present.

Parasitic Stomatitis.—As this disease is mainly due to lack of cleanliness during the lactation period, especially in bottle-fed babies, the chief remedy lies in the enforcement of scrupulous attention to the mouth, nipples, and feeding-bottles. The fungus is easily destroyed by bland antiseptics, such as glycerin or honey of borax. These should be gently rubbed on to the patches by the finger covered with a layer of gauze. A purge of calomel and rhubarb is also generally useful.

Aphthous Stomatitis is best treated by the local application to the ulcer, after it has been thoroughly dried, of solid silver nitrate or pure carbolic acid. Care should be taken to limit the application strictly to the ulcer.

Oral cleanliness should be enjoined, and a saturated watery solution of thymol used as a mouth wash is an excellent prophylactic antiseptic in the case of adults.

The dyspeptic or constitutional derangement is best dealt with by the administration of calomel and rhubarb or castor oil. This formula will be found effective :—

℞ Hydrargyri Subchloridi .... gr. ii
Pulveris Rhei .... gr. v
Pulveris Ipecacuanhæ .... gr. $\frac{1}{8}$
Sodii Bicarbonatis .... gr. iv
M

Ft. : Pulv : Mitte tales vi.

Signa.—One powder at bedtime, followed, if necessary, next morning by saline.

The exact dose can be modified to suit the age and the individual case. Later on, bitter tonics are helpful.

Ulcerative Stomatitis requires more drastic treatment than the two preceding forms. Probably the best method of dealing with it is by the application to the ulcerated surfaces of pure liquid carbolic acid. The small swab containing it should never hold any excess which might run beyond the sites of ulceration on to the sound mucosa. Tincture of iodine and silver nitrate may also be successfully employed.

The mouth should be washed frequently with thymol solution, or a weak solution of potassium permanganate, or peroxide of hydrogen. Aperients and tonics may also be indicated.

Glossitis.—The condition is met with in infectious diseases and digestive disturbances. It is usually easily cured when its cause is removed. Antiseptic mouth-washes, purgatives, and, later on, tonic treatment are successful in dealing with the derangement.

Salivary Glands.—The normal function of the salivary glands is important for the maintenance of a healthy digestion. A number of the diseases of these glands have no special bearing on digestive derangements. Only those which may cause disturbance of, or interference with, digestive function will be mentioned.

The importance of complete mastication and insalivation of solid foods, especially of bread-stuffs, has already been insisted on. In cases of amyl-dyspepsia, where no obvious cause for the condition can be ascertained, an examination of the mixed saliva should be undertaken to determine its diastatic activity and reaction. For this purpose a quantity can be collected in a beaker and the flow

may be facilitated by the aid of a little chewing-gum. The total amount of saliva secreted by the various glands is from two to three pints per diem. The activity of the glands can be stimulated in many ways. The mechanical act of chewing increases the output, while acids and pungent substances cause a large flow. Various psychic conditions may exercise an influence on the secretion. The thought of savoury food, of lemons or other acid fruits, suffices to cause an increased secretion. All irritations in the mouth, the various forms of stomatitis, bad teeth, and many inflammatory conditions, as well as the actions of some drugs—*e.g.*, pilocarpine—stimulate this secretion.

Increased salivation or *ptyalism* is a comparatively common complaint. Whether super-secretion of saliva is detrimental to gastric digestion is not yet definitely determined, but it can exist without causing any apparent derangement. This condition should be dealt with by ascertaining, when possible, its cause. Any obvious oral irritation should be removed, and atropine, in small doses, may be employed to restrict secretion should the salivation become embarrassing to the patient.

A diminution or hypo-secretion of saliva is less common. *Hypo-ptyalism* is probably associated with neuroses. Various mental disturbances, fear and emotion, may temporarily cause a dryness of the mouth. The continued fevers are often accompanied by a defective secretion and a dryness of the mouth which makes deglutition difficult.

The total suppression of oral and salivary secretion has been called by Hutchinson *Xerostomia* (dry-mouth). It is a rare affection. The entire buccal mucosa is affected, becomes dry, hard and shiny, and not infrequently fissured. Mastication, deglutition, and speaking are carried out with difficulty. This condition has probably a neurogenic origin, and is principally confined to women.

Atrophy of the salivary glands is a very rare disease. It may occur in old age in association with other senile changes. There can be little doubt that salivary failure is more compromising to the digestive function than super-secretion. Hypo-ptyalism and xerostomia should be treated by small doses of pilocarpine administered internally and by the use of sweets or pastilles containing small doses of citric acid. Chewing-gum may help to minimise these conditions by mechanically stimulating the glands. Galvanism may also be tried.

CHAPTER V.

THE ŒSOPHAGUS.

The physician should bear certain points in mind as to the course and relationship of the gullet.

It deviates twice from the middle line in its passage downwards. In the cervical and upper thoracic regions it bends to the left so as to project about a quarter of an inch beyond the trachea. At the level of the aorta it re-approaches the middle line, and then behind the pericardium it again bends to the left and forward to perforate the diaphragm and join the stomach obliquely. The œsophagus in its whole course largely conforms to the spinal curvatures.

Its length varies from eight to fourteen inches, averaging ten inches in measurement. From the central incisor teeth to the beginning of the gullet is six inches, so that from the lips to the stomach is about sixteen or seventeen inches in length.

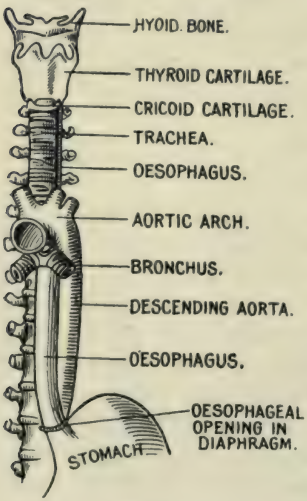


FIG. 12.
DIAGRAM SHOWING RELATIONS OF ŒSOPHAGUS.

The calibre of the gullet varies from half an inch, during collapse, to over one inch when distended. It opens obliquely into the stomach to the right of the fundus. Its junction with the stomach *anteriorly* corresponds with the seventh left costal cartilage at one inch from the sternum. *Posteriorly* it is at the level of the eleventh dorsal vertebra.

There are two narrowed portions or constrictions of the same size, flattened from before backwards. One is situated at the commencement of the tube, and the other where the left bronchus crosses the œsophagus.

The cardiac orifice of the gullet is still narrower in its lumen than the part immediately above. Here the muscular fibres are abundantly developed and, acting like a sphincter, prevent regurgitation. The bolus is always delayed at this point in its passage to the stomach for a time nearly as long as that occupied by the descent of the food down its entire length.

The gullet is a highly muscular canal composed of three coats, an external muscular, a middle or areolar and an internal or mucous. The muscular coat is composed of two layers, an external with fibres running longitudinally, and an internal with circular in the upper and oblique fibres in the lower part. These are continuous with certain muscular fibres of the stomach.

It is important to note that the epithelial lining is of the squamous type and, in primary cancer of the œsophagus, this type of tissue is reproduced in metastatic tumours.

Abnormalities of the Œsophagus.—Occasionally the gullet is the seat of abnormalities. It may become enormously dilated throughout its entire extent from Cardio-spasm, but this type of primary dilatation is rare.

Secondary dilatation of the gullet above the seat of any obstruction is far more commonly met with. In Carcinoma with stenosis the œsophagus becomes, in time, pouch-like above the seat of the constriction, and it may be so dilated that it becomes capable of holding considerable quantities of food which, when regurgitated, are apt to be mistaken for gastric contents which have been vomited. This error can be avoided by careful examination of the regurgitated material.

Of the other abnormalities the more important and better known are the Diverticula.

It is not quite certain whether they are acquired or congenital. The view has been advanced that diverticula result from a weakening in the tube due to failure of fusion of the lateral muscles posteriorly. The congenital weakness may permit of their formation.

There are two varieties, known as ' pressure ' and ' traction ' diverticula.

The traction diverticula are unimportant, and never cause signs of obstruction. They occur on the anterior wall of the œsophagus near the bifurcation of the trachea, and are most commonly produced by adhesions to tuberculous glands which have become the seat of periadenitis. The acts of coughing and breathing cause traction to be exerted on the gullet and produce the diverticulum.

Pressure diverticula are more important and of more common occurrence. They form only at the upper part of the œsophagus, and principally on the posterior wall. They are in all probability produced by the pressure of the bolus of food acting on a defective or weakened part of the muscle wall. Such diverticula are of various sizes and, at times, multiple. They are most common in men who are advanced in years and, until they attain considerable size, may give rise to no symptoms. When they become distended with food they may cause dysphagia and some respiratory difficulty. Occasionally they may rupture into the thoracic cavity and produce pulmonary gangrene or abscess of the lung.

Symptoms and Signs are rarely present unless the diverticula attain considerable size. The smaller ones are usually discovered accidentally or *post mortem*.

When the larger diverticula become distended with food, a sense of obstruction under the sternum, with some dysphagia, or even some respiratory difficulty, may be experienced.

If regurgitations of undigested food, found on examination to contain no gastric juice, are of frequent occurrence, the possibility of this condition should be borne in mind. The retention of food may produce a foul breath and inflammatory changes in the œsophagus with pain along its tract, aggravated by the act of deglutition.

The stomach tube or the olive sound usually passes over the orifice of a small diverticulum; but from time to time the point of the tube may pass into the sac, preventing its further passage downwards. If so arrested, aspiration should be resorted to in order to determine the presence or absence of food. The greatest care is required in the use of stomach tube or olive sound, as the sac may be ruptured by any undue pressure. Should there be any doubt as to the condition, X-ray and opaque meal may be employed to further the diagnosis. It should be noted, however, that this method is not always successful, and may be misleading, for if the saccular dilatation be already full of food the whole of the opaque meal may pass over the orifice without entering and no shadow be obtainable. The œsophagascope may also be employed for diagnosis.

Treatment.—For those cases discovered accidentally no treatment is required. When, however, dysphagia, pain, regurgitations or respiratory embarrassment result, surgical interference may be

necessary. Rarely is obstruction so complete as to necessitate a preliminary gastrostomy for the sustenance of the patient. If a stomach tube can be introduced into the stomach, in those cases where obstruction interferes with swallowing, food can be administered in this way morning and evening.

A rarer abnormality is a *doubling of the tube* in some part of its course, and still more rare is a communication between the trachea and the œsophagus—a condition incompatible with life.

It will be obvious that in many conditions where the use of the stomach tube may be indicated these anatomical relationships and aberrations of the œsophagus must be kept in mind.

Structural alterations in the gullet will usually be revealed by the stomach tube, sound, or œsophagoscope, while information regarding the extent, situation, and degree of constriction, dilatation, or other abnormality may also be demonstrated by radiography.

Examination of the Gullet.—Not infrequently the gullet requires special examination for some disease, and wherever the stomach tube has to be passed incidentally an examination of the œsophagus is made.

The more ordinary methods of physical diagnosis are not available for examination of the œsophagus. It is true that in the neck it may be palpated on the left side behind the trachea. A tumour or a distended diverticulum in this region may be detected by the fingers.

Auscultation of the œsophagus does not yield very reliable information. The whole act of deglutition is too rapid to be well observed. The murmur or sound of swallowing can be best heard in the neck on the left side of the trachea. In the thorax the sound is most audible to the left of the spinal column at the level of the seventh dorsal vertebra. The swallow sound can also be heard at the xiphoid cartilage.

The stethoscope is placed posteriorly at the seventh dorsal vertebra and the patient asked to swallow some fluid. There is at first, at the moment of swallowing, the sound of deglutition, then, in six or seven seconds later, the "œsophageal bruit," and still a few seconds later the sound produced by the fluid entering the stomach. Delay or suppression of the œsophageal bruit, or of the sound produced by the fluid entering the stomach, is suggestive of partial or complete stenosis of the œsophagus.

A more reliable auscultatory sign is to observe the effect of respirations on the deglutition act without taking more fluid. As has been pointed out by Revidtzev, in all marked œsophageal obstructions there is a retention of fluid, for a varying time, in the pouch-like dilatation immediately above the site of stricture, except in cases where the stricture is almost impervious. Each act of deglutition causes a fresh descent of fluid and a complete repetition of the sounds, indicating that some of the originally swallowed fluid has been retained in the œsophagus and that therefore obstruction exists.

More recently the œsophagoscope has been employed to examine the gullet and stomach. The use of this instrument requires considerable technical skill, and its employment causes great discomfort, if not actually some risk, to the patient, even though anæsthesia be employed. From what I have seen of œsophagoscopy I am not convinced that the information gained from its use is superior to that obtained by the more simple, safe, and easy methods in common use; but there are certain cases in which this method of investigation may be necessary. If a diagnosis fails to be made by all the other methods of investigation the œsophagoscope may be used.

The œsophageal bougie and stomach tube yield, in competent hands, a great deal of valuable information. In most instances it suffices and is safer to employ the soft stomach tube rather than the harder bougie. If a solid bougie be chosen for examination of the gullet, it should be flexible and perfectly smooth. It should be steeped in hot water to soften it a little, and then smeared with butter, vaseline, or glycerin. The too frequent use of oil as lubricant rapidly deteriorates the rubber and shortens the life of the tube.

With the patient in a sitting posture the bougie or tube is held in the hand like a pencil and passed directly on to the posterior wall of the pharynx through the widely opened mouth. There is no risk of it entering the larynx if its point is passed down the pharynx. If the patient is very nervous, or the pharynx very irritable, and much reflex coughing and spasm be set up, the mucous membrane can be sprayed with a weak solution of cocaine, and in a few minutes the difficulty will pass over. The solid flexible bougie is perhaps more adapted for palpating the œsophagus and estimating the presence, extent, consistence, and situation of

obstructions or other abnormalities. Different sized bougies may be employed to estimate the degree of stenosis.

The Olive Bougie is the better instrument for examination of the gullet. A moderately large sized olive should be used first, and if the operator fails to pass it into the stomach, smaller olives may be substituted. No force must be used during such an examination, and no severe pain should be evoked. If any is complained of during the descent of the bougie, it should be located by measuring on the bougie the distance at which it occurs from the incisor teeth. On withdrawing the bougie or tube it should be carefully examined for the presence of blood, cells, or fragments of tissue. These should be subjected to microscopic examination, more particularly if malignant stenosis be suspected.

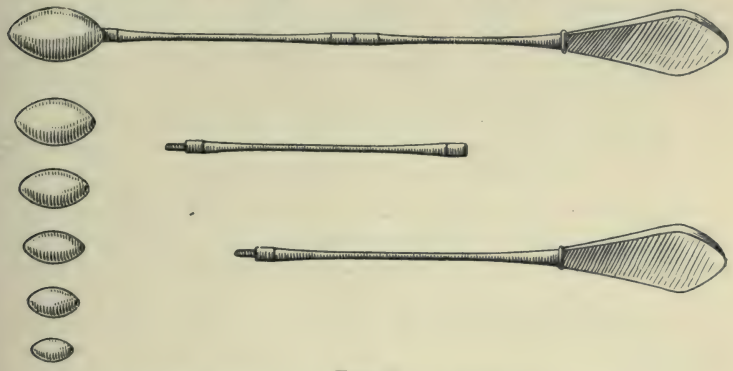


Fig. 13.
Einhorn's Divisible Œsophageal Bougie and Olives.

By means of the olive bougie an obstruction may be accurately palpated and the degree of stenosis, as well as its length, can sometimes be accurately measured. The exact distance down at which the obstruction begins should be measured on the stem. Very gentle pressure should then be employed. If difficulty be experienced in passing an olive of a given size the instrument should be withdrawn and a smaller olive adjusted. When the obstruction is cleared, and the olive liberated below it, the bougie should be gently withdrawn, and the exact point at which it re-engages in the constriction once more measured on the stem. The situation, degree, and length of the obstruction will then be fairly accurately defined. In many instances the stricture will be found

to be impermeable to even the smallest sound. If malignant disease be excluded, the apparent impermeability of simple stenosis may sometimes be overcome by the silk thread method of dilatation (*q.v.*).

Radioscopy affords reliable information in cases of stenosis, and sometimes reveals the presence of pressure diverticula. The position, extent, and degree of the constriction can be seen on the screen by the massing of the opaque bolus immediately above the narrowing, and by the thin line produced by the trickling of the barium or bismuth through the stenosed portion. Diverticula can be located, when the opaque substance enters them, by the stationary shadow cast on the screen.

If the stomach tube be used, and it appears to pass into a pressure diverticulum, it is useful, while it is held in its retained position, to employ gentle aspiration. In this way, if food *debris* be present, it can be extracted, and the diagnosis verified. In some cases I have been able by this means to demonstrate that suspected malignant obstructions were nothing more than diverticula.

Diseases of the Œsophagus.

Œsophagitis.—*Acute* inflammation is rarely primary, but results from the local action of irritant and corrosive poisons or from severe septic conditions associated with certain fevers. It is more or less present in cancer of the gullet.

The Symptoms are pain and dysphagia. The pain is usually substernal.

Treatment depends on the condition which has produced the œsophagitis. A few drops of olive oil placed on the tongue and slowly swallowed sometimes affords relief, and a sedative antiseptic will be found in glycerin of borax administered in the same manner as the oil. A pastille containing chlorate of potash and cocaine is also useful. Chips of ice are always grateful.

Chronic Œsophagitis may ensue from any of the causes giving rise to the acute form. The most important type of chronic œsophagitis is that which arises in alcoholics with organic changes in the cardio-vascular and hepatic systems. When the free return of the portal circulation is impeded in chronic heart disease, and more particularly in cirrhosis of the liver, a collateral circulation is produced in which the œsophageal veins play an important rôle. These become distended, varicose, and frequently rupture into the

œsophagus, giving rise to one of the commonest forms of hæmatemisis associated with Laennec's cirrhosis of the liver.

Symptoms of chronic œsophagitis are not well-defined, and often are overlooked because dominated by those of the more important disease which produces the abnormality in the gullet. Some dysphagia and sternal pain, aggravated by swallowing, may be the only complaint. Pungent substances and alcohol are specially prone to evoke the symptoms.

Ulcers of the Œsophagus are not common. There are two types, simple and malignant.

Simple ulcers are often not detected during life, and may give rise to no diagnostic symptoms. They occur in the lower part of the gullet, close to the cardia, and within the range of action of gastric juice. Their formation is favoured by any condition which permits of regurgitation from the stomach, such as cardiospasm. When pain and dysphagia are symptoms and ulcer of the œsophagus suspected, its presence may sometimes be confirmed by means of a thick white silk thread, which should be swallowed and be long enough to pass well into the stomach. The thread should be attached to one of the incisor teeth, and left *in situ* for some time before being withdrawn. The existence and position of an ulcer may be demonstrated by a bloodstain on the thread.

Œsophageal ulcers may perforate into the thorax or cause subsequent constriction.

Their treatment is similar to that of gastric ulcer.

Malignant ulcer of the gullet is merely a sequel and an accompaniment of stenosis due to the new growth.

Foreign Bodies in the Œsophagus.—Neurotic subjects sometimes assert that ' something ' is sticking in the gullet. Such sensations are not produced by an actual foreign body, but have the same origin as the *globus hystericus*. Portions of bone, coins, buttons, etc., are frequently swallowed, especially by children. They may become impacted and a source of pain and danger. If we know that the body is small and smooth, it may safely be pushed into the stomach. This should not be attempted if we are in doubt about the nature and position of the foreign body. X-rays should be used in all cases where a shadow would be produced. Surgical aid may have to be invoked for the removal of certain impactions.

Spasm of the Œsophagus, causing stricture, is unimportant. It is an incident in certain cases of hysteria and other neuroses. The spasm may occur at any part of the gullet. An interesting form, interfering with the entrance of food into the stomach, is the neurosis, probably due to irritation of the vagus, known as *cardiospasm*. The obstruction is intermittent. The clamped tube or bougie will for a time refuse to pass on, and then relaxation follows, with liberation of the tube. This condition will be more fully discussed under the motor derangements of the stomach.

Stricture of the Œsophagus is by far the most important lesion. It may be *simple* or *malignant*. It is obviously important for prognosis and treatment to be able to differentiate these two conditions.

Obstructions of the gullet may also occur from disease outside the tube, as enlarged glands, new growths, and aneurismal tumours.

Sometimes stenosis of the œsophagus may be congenital.

Simple Stricture.—By far the most common form is that which results from the action of corrosive poisons, the sequel of ulceration. The stricture is formed by fibrous or scar tissue. Such strictures are most apt to be found at those parts of the gullet which are narrowest, and where delay is caused in the downward passage of the corrosive. The amount of stenosis present will determine the character of the symptoms. When obstruction is nearly complete, no solid and only small quantities of fluid foods can be swallowed. There will be, in these cases, a continuous regurgitation, after a varying time, depending on the amount of dilatation which forms above the stricture. If a small stomach-like pouch is formed in the gullet the regurgitation is considerably delayed after the act of ingestion. There need be no confusion between such regurgitation of food and real stomachic vomiting. The absence of any digestive action on the food, which will be returned little changed, and the entire absence of free hydrochloric acid and pepsin can be demonstrated on testing.

There is usually a history of a progressive and slow development in the dysphagia and an absence of pain and cachexia. The auscultation sound of the food passing through the cardia is absent. The passage of the stomach tube, and the employment of X-rays and an opaque meal or bolus will demonstrate the nature of the condition.

Treatment of such cases depends on the degree of stenosis present. The food should be fluid, and taken frequently in small doses. The amount and frequency of the feeding prescribed will depend on the nutrition of the patient.

The constriction may be slowly dilated by the daily passage of graduated bougies.

In attempting to dilate a fibrous stricture of the gullet we are sometimes met by the initial difficulty of apparent impermeability to the sound. Before accepting failure to permeate the stricture Plummer's silk thread method and hollow bougie should have a trial. The patient is directed to swallow three yards of surgical thick silk thread. The descent is aided by sipping some warm fluid. Next day another three yards are similarly swallowed. Few strictures are so tight as to be impermeable to the thread. A very long portion of the thread finds its way, by peristaltic action, through the pylorus and down into the intestine, where it becomes sufficiently fixed to resist some traction.

Over this thread, which acts as a guide, a hollow bougie, ending in a perforated olive, is passed, which frequently can thus be made to find its way through the stricture. No undue force should be employed. Once the stenosis has been permeated, the daily use of the bougie with larger olives will, in time, accomplish a full dilatation.

If the fibrous stenosis be non-dilatable, and if the patient is suffering from serious and progressive malnutrition, surgical interference may be considered. Unfortunately in those cases where surgical procedure is most called for it is often least possible ; but a gastrostomy may be undertaken as a means of maintaining the patient's strength and prolonging life.

Abbe's ' string cutting ' method of dealing with simple permeable strictures is worth a patient trial. The patient is nourished through a flexible catheter, passed through a small gastric fistula, till dilatation of the stricture is accomplished and normal deglutition restored, when the fistula is then closed.

Malignant Stenosis.—Carcinoma is the most common disease of the œsophagus, and it occurs most frequently in its lower half. The type of carcinoma is that of the squamous-celled epithelioma. Much more rarely it takes origin not in the mucous membrane, but in the glandular tissue in the proximity of the œsophagus,

and is in type an adeno-carcinoma. It may spread by infiltration longitudinally or circumferentially. It causes almost invariably a progressive stenosis of comparatively rapid development. As constriction takes place, dilatation occurs above the seat of the obstruction, and sooner or later ulceration may supervene. Many important structures may become involved. Pressure symptoms may become urgent, from implication of the bronchus, pleura, lung, or aorta. The recurrent laryngeal nerves are usually found to be implicated, and, when the growth is extensive, there may be erosion of the vertebral bodies. Perforation of the ulcer may take place into any of the above structures and produce acute symptoms going on to a fatal termination.

The Diagnosis of cancer of the gullet need give rise to no serious difficulty. Substernal pain, real dysphagia, regurgitation of food, emaciation, cachexia, and loss of appetite form, in a man of forty years or upwards, a picture that is very suggestive. Frequently large quantities of glary mucus are constantly regurgitated, and give rise to much annoyance and distress to the patient. The stomach tube or bougie, with which we may demonstrate the existence of stenosis, must be used with the utmost caution in all cases of suspected malignant disease. Any forceful manipulation may result in perforation of the friable tissue. Later on, when ulceration begins, the breath may become fœtid, and the regurgitated material may be contaminated with blood and pus, or fragments of tumour tissue.

If there be much infiltration into the surrounding parts, and pressure be great, pain may be severe and constant, and may radiate to parts remote from the mediastinum. Other signs and symptoms may shortly develop. The lymphatic vessels pass to the inferior set of deep cervical glands. The cervical and supraclavicular glands should in all such cases be frequently under observation, more particularly those on the left side, for signs of involvement by new growth.

From extension of the carcinoma into the veins there may be a general metastatic deposition in the skin and elsewhere.

Commonly, though not always, in the earlier stages of the disease free hydrochloric acid gradually diminishes and ultimately disappears from the gastric juice. Later on, when the nutrition becomes seriously impaired, acidosis may appear and acetone may be found in the urine.

The disease terminates fatally from exhaustion, hæmorrhage, or perforation, usually within a year from its commencement.

There may be some difficulty, in certain cases, in the differential diagnosis of œsophageal cancer from intra-thoracic tumours (glands, aneurism, mediastinal growths). A careful examination of the intra-thoracic organs, the passage of a soft bougie, and the employment of radiography will in doubtful cases clear up the diagnosis.

The Treatment must be largely symptomatic. A strictly fluid diet of concentrated nourishment, rectal salines, sedatives if pain be great, are all useful, and, before malnutrition becomes extreme, gastrostomy may be safely performed. It gives great temporary relief to the patient. If achylia has been ascertained to be present, an artificial digestive containing dilute hydrochloric acid and glycerin of pepsin is useful in promoting digestion and appetite.

Syphilitic and Tuberculous Affections of the Gullet are exceedingly rare.

CHAPTER VI.

PHYSICAL EXAMINATION OF THE ABDOMEN.

Every physician should have an accurate notion of the topographic areas of the abdomen. No case of digestive derangement should be dismissed without a careful examination of the abdomen generally.

The bony prominences, the umbilicus and the lineæ, constitute the landmarks for the regional divisions. It should be noted that the umbilicus, as the nipple, is somewhat variable in position. In each of the regions lie certain organs, or parts of organs, and it is convenient in case-taking to refer to the regions when abnormal conditions are found in any one of them. The abdomen and its contents should be examined first when the patient lies in the dorsal decubitus; but valuable information can often be obtained by examination of the abdomen in many other positions—*e.g.*, sitting or standing. The various methods usually employed in examination of the thorax are applicable to the abdomen.

(1) **Inspection.**—The first thing to note is any superficial alterations and the condition of the skin. A glossy smooth skin usually indicates some intra-abdominal pressure with over-distension. This condition is often well seen in a massive ascites.

During pregnancy the median line becomes deeply pigmented, particularly in women of dark complexion. Icteric discolouration is usually well marked over the abdomen, as is also the pigmentation of Addison's disease. Absorption lines—Lineæ Albicantiæ—formed from prolonged distension, may afford evidence of past ailments. It ought to be borne in mind that such striæ are frequently found, in quite young women, in the lumbar, iliac, and hypogastric regions, who have never been pregnant nor suffered from any abdominal disease. They may be regarded as due to a too rapid absorption of fat in these situations. Various rashes are often seen on the

abdominal wall. The pruriginoid eruption associated with phthiariasis and scabies is here prominently developed. The abdominal skin is the principal seat of the enteric eruption, which comes out in successive crops. Hæmorrhagic spots are of common occurrence in the abdominal wall in many blood diseases, septic conditions, and from the bites of vermin.

Any old scars, especially in the groin, should be noted, and their exact causation ascertained.

Abnormal vessels on the abdominal wall require careful scrutiny. If enlarged veins be associated with ascites, a proper study of the vessels involved, and the direction of the blood stream flowing through them, may greatly aid the diagnosis of the intra-abdominal condition. If we have gross obstruction of the inferior vena cava or iliac vein from tumour pressure or from thrombosis, the distended vessels occupy the sides of the abdominal wall, and the blood flows through them from below upwards. The lateral collateral circulation established in such cases is due to the obstruction of the inferior vena cava and the distension of the superficial veins conducts the blood by the abdominal wall to the superior vena cava.

In obstruction of the portal circulation the dilated veins are principally on the median line, and often radiate from the umbilicus, forming the 'Caput Medusæ.' This type of venous distension is only found in cirrhosis and thrombosis of the portal circulation.

The size of the abdomen with reference to the body generally should be noted. Any generalised or local enlargement will have to be registered. Inspection should be carried out from the front, sides, and back in the recumbent, sitting, and erect postures.

Enlargements of the abdomen are commonly due to one of four causes :—

(*a*) *Fat.*—Obesity often shows itself more prominently here than elsewhere. It should never be mistaken for any intra-abdominal condition. The picture it presents is easy of recognition. The uniform projecting enlargement, the dimpling on the surface, showing its distribution in areas bound in by connective tissue, the depression of the umbilicus, and the probable good nutrition of the rest of the body generally makes the diagnosis easy. At times *œdema of the abdominal wall*, part of a general anasarca, may give rise to an appearance which is not unlike that due to obesity ; but a further examination will easily differentiate these conditions.

(*b*) *Gaseous Distension.*—Gas may be free in the peritoneal cavity or inside the hollow viscera. Free air in the cavity is due to perforation, and may form part of the picture of acute peritonitis after rupture of gastric or appendical ulcers. There is a uniform distension of the cavity, and it may become impossible to localise the solid viscera. The loss of hepatic dulness is somewhat characteristic.

In tympanitis or meteorism due to general distension of the intestine from gaseous accumulation the relative positions of the organs are usually able to be made out. The individual coils of the gut can often easily be seen. When meteorism exists, and the coils of the intestine can be seen, peristaltic movements ought to be looked for, and their sites of origin and the direction of the waves carefully noted.

Visible peristalsis is usually associated with some form of intestinal obstruction. Much more rarely it is a manifestation of neurasthenia or hysterical condition.

In pyloric obstruction the outline of the distended stomach can frequently be demarcated in the epigastric region, and visible peristaltic waves may be seen travelling from left to right. If the transverse colon be the seat of stenosis or obstruction, the waves run in the opposite direction—from right to left.

An obstruction near the ileo-cæcal valve causes the central part of the abdomen to become more prominent, and here the coils of the bowel are seen lying on the top of each other, giving rise to the so-called ' step-ladder ' formation.

An obstruction low down in the colon may produce a horse-shoe distension, due to generalised inflation of the colon, round the circumference of the abdomen.

Local distensions of any other part of the bowel—slowly rising up and showing active peristalsis with sudden collapse, accompanied by loud gurgling—is to be interpreted as a sign of partial occlusion at the site of the distension.

(*c*) *Free Fluid* in the peritoneal sac (ascites) produces a characteristic uniform distension of the abdomen. In the dorsal decubitus the gravitation of the fluid to the sides produces lateral bulging. When the effusion is not too great, the distension of the flanks may, in the dorsal decubitus, produce a degree of flattening of the umbilical region, and a sudden change of position of the patient may produce a sudden distension of the dependent portion of the abdomen. Further and conclusive evidence of the presence of

free fluid can be obtained by palpation, percussion, and paracentesis.

I have seen dilatation of the stomach, with retention of its contents, so extreme as to produce many of the physical signs of ascites. It should be noted that a succussion sound is never heard from ascites, while a loud splashing sound is almost invariably present in those cases of extreme dilation. Should any doubt exist, the stomach should be emptied by the syphon and the abdomen again examined, when all confusing signs, due to dilatation and retention, will forthwith disappear.

(*d*) *Tumours.*—Abdominal distension is invariable from all tumours of considerable size. Small masses often escape detection because they produce no visible alteration. Tumours and cysts usually produce distensions which are not uniform, and which are localised to certain regions. Tumours of some size displace the hollow viscera, and so give rise to a further asymmetry. The maximum distension and resistance is often in the middle line.

Pregnancy and over-distension of the bladder must always be excluded. The characteristic signs of gestation will prevent an error as regards pregnancy, while any doubt about a suprapubic tumour being a distended bladder can be removed by passing a catheter.

For the differential diagnosis of tumour many methods of investigation may be required.

Enteroptosis.—This condition can often be recognised by inspection alone—especially when carried out with the patient in different postures. It consists in undue relaxation of the abdominal wall, resulting from frequent pregnancies and the wearing of tight corsets, while neurasthenic and debilitating conditions may also be responsible for its production. The loss of supporting intra-abdominal fat is a potent factor in the production of this condition. In addition to the relaxed parieties there is general relaxation of the mesentery and parietal peritoneum. The viscera become unduly movable. When all are implicated, we speak of the condition as 'Glénard's Disease,' visceroptosis, gastroptosis, or splanchnoptosis. The appearance of such an abdomen shows relaxed and wrinkled walls and a general flabbiness, which is quite abnormal. The contour of certain viscera, such as the stomach and colon, often become visible. The abdomen seems, so to speak, to flow over the bed, projecting at each flank and being flat in front.

In the erect position the viscera fall downwards, and give rise to a flattened appearance in the epigastric and to undue prominence in the hypogastric region.

(2.) **Palpation.**—Abdominal palpation is very important in every case where abdominal symptoms are present. It verifies or disproves the results of inspection. To yield reliable data it must be done thoroughly, and certain points in the technique must be carefully kept in mind. The abdomen is generally very sensitive, and the reflexes usually are active. Any roughness or maladroitness will defeat the object of the examiner. The entire abdomen should be exposed, and the physician should place himself at the right-hand side of the couch or bed. The hand should be thoroughly warm—excessive cold being extremely disagreeable to the patient. The first part of the examination should be undertaken in the dorsal decubitus, with the head on a low pillow. The patient should breathe quietly, and his attention be diverted by engaging him in conversation. Palpation should be made with a smooth, gliding, rotatory movement, and sudden jerking and pressure with the tips of the fingers avoided. The pressure, at first light, may be gradually increased till the palpating fingers feel deeply into the abdomen. The presence of tenderness can best be elicited by the facial expression of the patient. It should be remembered that deep pressure over the epigastric and upper parts of the lumbar regions evokes pain in many normal subjects. In hysterical or neurotic people, when pain is complained of in a definite area, palpation should be made over another area at the same time as over the site of the pain. It will thus often be found that there is no more real tenderness in one part than in another. It is always best to start palpating in the most insensitive parts of the abdomen, and away from those areas in which the pain is located. Palpation should be carried out with the patient in several positions. Sometimes, if very deep palpation be desired, the two hands may be employed—the one on the top of the other—to reinforce the pressure. Palpation not infrequently requires to be bi-manual. Wherever the bony skeleton is absent, as in the lower margins of the hypochondriac and lumbar regions, the abdominal contents in these situations can be compressed between the two hands. Deep tumours and movable kidneys, which might not be felt by the single hand, are readily detected by this bi-manual method.

All palpations of the upper segment of the abdomen should be carried out both during quiet and forced respiration. Any mass lying in close proximity to the diaphragm will descend during inspiration, and becomes easy of detection.

When there is fluid in the peritoneal sac, the ordinary method of steady firm palpation may not suffice to feel the underlying parts. In such cases, when the abdominal wall is carried away from the deeper organs—for example, the liver—by fluid, ' dipping ' palpation with the finger tips, made by a sudden thrust inwards, displaces the fluid and brings the finger on to the organ or the tumour.

In palpation, then, the object is to obtain the greatest relaxation of the abdominal wall. In some instances this can be facilitated by flexion of the knees ; but if, in spite of all manipulatory expedients, sufficient relaxation cannot be obtained, it is certain that no examination of the abdomen, yielding negative results, is reliable. In all cases, therefore, where rigidity cannot be overcome and doubt remains, an anæsthetic should be administered.

In the first instance the digestive tract should be empty, and the examination made on a fasting stomach and after purgation. Many a scybalous mass has been, with unfortunate results to the physician, diagnosed as a tumour for want of these precautions. Care must be also exercised when palpating the abdomen not to confuse an actively pulsatile aorta for an abdominal aneurism, or to fall into the error of mistaking a tumour, which overlies the aorta and pulsates with it, for an aneurism.

Special forms of palpation are employed for the diagnosis of fluid in the abdomen—ascites or cystic tumours—and for the recognition of œdema in the wall, fluid in the colon and gastric retention, as well as for other purposes.

Ascites.—The most characteristic sign of ascites is the fluctuation wave. This consists in the appreciation by the palpating hand, placed on one side of the flank, of the short vibration thrill transmitted through fluid, when the opposite side is lightly but sharply tapped with the tips of the fingers of the other hand. Great obesity or œdema of the wall may give rise to a pseudo-fluctuation, which may lead the inexperienced into error. The ulnar side of an assistant's hand placed in the middle line of the abdomen cuts off such vibratory waves, and no transmission takes place to the observer's hand. If further proof of the presence of free fluid in

the peritoneum be desired, the results of percussion will demonstrate its presence.

Large cystic tumours in the abdomen give rise to local bulgings over which fluctuation can often be detected by the palpating hand placed at one pole, while the other pole of the tumour is sharply struck by the finger tips. If the other methods of investigation be employed, such a tumour can hardly be mistaken for ascites.

Œdema of the abdominal wall is easily demonstrated by pinching up a portion of the skin or by sustained pressure of the finger.

The detection of movable or floating kidneys and of abnormal amounts of fluid in the stomach is so important that it will be considered later on.

Palpation is very important as a means of detecting local increased resistance in the abdominal wall. A general resistance and rigidity found almost universally over the abdominal wall is characteristic of a general peritonitis; but the examiner is much more apt to overlook the localised spasms of underlying muscles. The regions in the abdomen requiring to be specially examined for increased rigidity are McBurney's point for appendicitis and the upper bellies of the recti, for the localisation of gastric, duodenal ulcer and gall bladder affections. A marked localised rigidity in these parts is of great significance, indicating an attempt on nature's part to throw the overlying wall into spasm as a protective act.

(3.) **Abdominal Percussion.**—The percussion of the abdomen, except over the hepatic and splenic areas, yields generally a tympanitic note. The note varies in different patients and at different parts, according to the size of the air-filled organ and the amount of its gaseous, liquid, or solid contents. The full and tonic stomach and the full colon give rise to a dull note, and in constipation, with fœcal accumulation, there is usually a dull note over the sigmoid flexure. Ordinary superficial percussion is of doubtful value except for the delimitation of the stomach and colon when they are sufficiently distended. The normal tympanicity of the abdomen may be completely lost in great obesity, in very massive ascites, and in tumours which occupy the entire cavity or where there is greatly thickened peritoneum.

Light percussion usually yields more accurate results than deep percussion. Indeed, a strong stroke may fail to elicit slight superficial dulness—*e.g.*, over the lower border of the liver.

There are several special methods of employing percussion in abdominal work which have reference to the demarcation of the different organs. That of auscultatory percussion (including 'scratching percussion') is so important in the delimitation of the stomach that it will be fully described in a later section. The use of the tuning fork in differentiating solid from air-filled organs can often be usefully employed.

(4.) **Auscultation** may be used for the detection of friction over solid organs which move on respiration. Peri-hepatitis and peri splenitis may be so discovered.

Auscultatory percussion to delimitate the stomach may also be of value. (See examination of the stomach, page 85)

CHAPTER VII.

TOPOGRAPHIC RELATIONS OF THE ABDOMINAL ORGANS.

Solid Viscera.—No attempt can be made to describe at any length the anatomy of the abdomen. Only those organs which most frequently require to be examined may be dealt with in some detail.

Many abdominal symptoms are common to several diseases, and no examination can be complete without a systematic scrutiny of each of the organs concerned in gastro-intestinal functions.

It is essential to have a clear conception of the relationship of the organs to each other and to the surface of the body.

The Liver.—This organ is almost entirely protected by the skeletal structure. The main bulk of the liver lies in the right hypochondriac region (right lobe), and the remainder in the epigastric region (left lobe), and slightly in the left hypochondriac region as far as the left mammary line. In this situation it occupies the entire antero-posterior diameter of the abdominal cavity. In the erect posture the liver is about half an inch lower than in the recumbent.

The dome-shaped upper surface of the liver is in close contact with the vault of the diaphragm. It is therefore freely movable with respiration. It is readily displaced by disease in the neighbouring parts and varies greatly in size according to the amount of blood it contains.

The *upper border of the left lobe* corresponds to a transverse line drawn through the lower end of the meso-sternum, while the *upper border of the right lobe* in the mammary line is at the level of the lower border of the fifth rib ; in the mid-axillary line it is at the level of the seventh, and at the mid-scapular line at the level of the ninth rib.

The *lower border posteriorly* is opposite the eleventh spinal process, and at the mid-axillary line it corresponds to the costal margin. *In front* the lower border, between the mid-axillary line and the right linea semilunaris, lies about half an inch below the costal margin, and then crosses the epigastrium between the umbilicus and the end of the ensiform.

The *under surface* of the liver comes in contact with the stomach, duodenum, gall-bladder, transverse colon, and small bowel. Anteriorly, posteriorly, and laterally, the liver is in contact with the parieties.

It is not possible to determine with accuracy the upper border of the liver by our usual methods of physical diagnosis, because it lies so deeply. The best method of ascertaining the position of the upper margin of the liver is to map out the diaphragmatic attachments. It will vary with, and correspond to, the position of the diaphragm. It is therefore of far greater importance to delimit its lower border than to attempt to ascertain the position of its upper surface. Normally the lower border can be vaguely felt in the mammary line when the abdominal wall is sufficiently relaxed. If it descends in this region so as to be easily palpable it is probably slightly enlarged or displaced.

The liver should be examined by the following methods :—

(*a*) Inspection, either in the erect position or in the dorsal decubitus, often shows an enlarged liver moving freely below the costal arch. Sometimes through a thin abdominal wall tumours on its surface can readily be seen projecting.

(*b*) Palpation, in the dorsal decubitus, with knees flexed, yields the most valuable data. The methods of palpation already described for general abdominal work are to be employed. The edge of the liver should be examined for its consistence, form (sharp or blunt), and outline. The surface should also be examined in the same way to ascertain the presence of tumours or other irregularities. In all cases the organ should be examined both during quiet and forced inspiration, and the ' notch ' at the gall bladder should, if possible, be located. The normal gall bladder cannot be palpated, but if distended it forms a pear-shaped smooth tumour. During palpation of the liver the presence or absence of local or general tenderness will be noted. The liver may be rotated, and appear enlarged, by the presence of a tumour behind it.

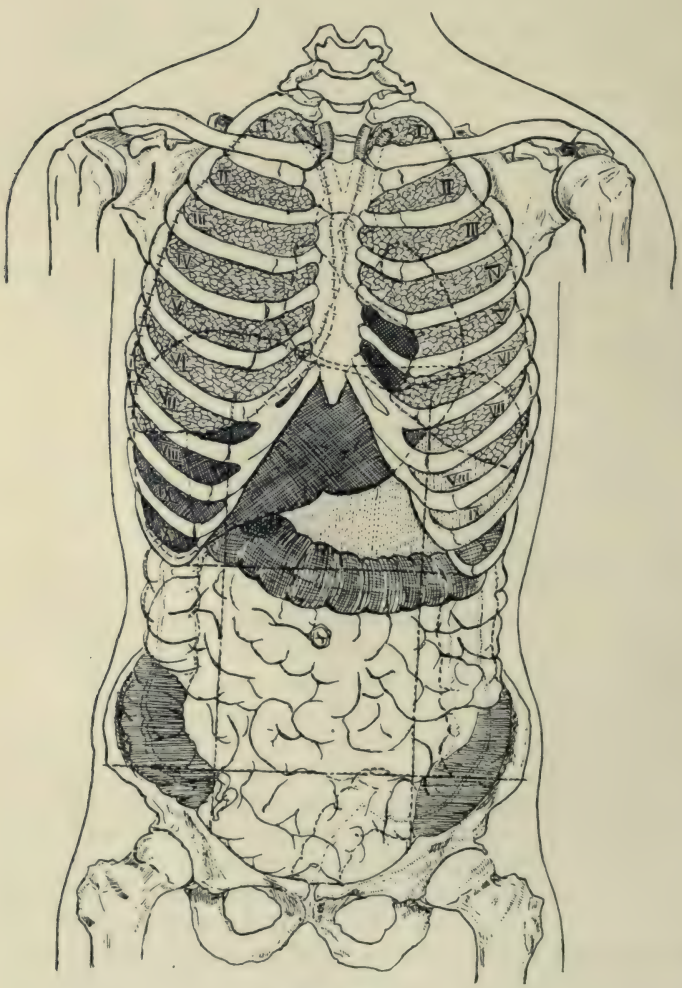


FIG. 14.

DIAGRAM OF ABDOMINAL VISCERA AND LUNGS, ANTERIOR VIEW (after Deaver).

ABDOMINAL REGIONS AND THEIR ANATOMICAL CONTENTS

(From Deaver's Surgical Anatomy, Vol. III, *slightly modified*).

Right Hypochondriac.	*Epigastric.*	*Left Hypochondriac.*
Liver (part of right lobe).	Liver (left lobe, quadrate, caudate, and Spegelian lobes).	Liver (small portion of left lobe occasionally).
Gall Bladder (divided by longitudinal line)	Gall-Bladder (divided by longitudinal line).	Spleen.
Kidney (upper and outer parts).	Stomach (pyloric and middle portions with cardiac and pyloric orifices).	Pancreas (tail).
Colon (hepatic flexure and part of ascending colon).	Intestine (first, second, and fourth portions, and termination of third portion of duodenum, and part of the transverse colon).	Kidney (upper and outer parts).
	Pancreas (head and body).	Stomach (cardiac end).
	Kidneys (upper and inner parts, with sinus and pelvis of the ureters.)	Colon (splenic flexure and part of the descending colon).
	Suprarenal Bodies.	
	Spleen (upper and inner parts).	

Longitudinal plane at outer border of left rectus muscle.

Upper transverse plane at level of the lowest point in costal border.

Right Lumbar.	*Umbilical.*	*Left Lumbar.*
Kidney (lower and outer parts).	Kidneys (lower inner portion).	Kidney (lower and outer parts).
Intestine (ascending colon, part or all of cæcum, and part or all of appendix. Some of the small intestine, mostly ileum).	Ureters.	Intestine (small intestine, mostly jejunum; descending colon and part of the sigmoid flexure).
	Intestine (lowest part of the third portion of duodenum; part of the jejunum ileum and transverse colon; and, usually, part of the sigmoid flexure).	
	Uterus in Pregnancy.	

Longitudinal plane at outer border of right rectus muscle.

Lower transverse plane through anterior superior iliac spines.

Right Iliac.	*Hypogastric.*	*Left Iliac.*
Intestine (small intestine, mostly ileum; sometimes the tip of the cæcum and part or all of the appendix).	Intestine (jejunum and ileum of small intestine and part of the sigmoid flexure).	Intestine (small intestine and part of the sigmoid flexure).
	Ureters.	
	Bladder (in children and, when distended, in adults).	
	Uterus in Pregnancy.	

(*c*) Percussion.—It will be borne in mind that the upper part of the right lobe is overlapped by lung tissue. Over this area there is relative hepatic dulness. The parts in contact with the abdominal wall give absolute dulness. This absolute dulness should be made out anteriorly, laterally, and posteriorly, by percussion in the mammary, mid-axillary, and scapular lines. The percussion should be from above downwards for the upper border, and for the delimitation of the lower border from below upwards. Strong percussion may be employed to determine the upper, and very light for the lower, border of the liver. The lower border in the mammary line corresponds generally with the costal arch. Here the vertical extent of hepatic dulness is normally four inches, in the mid-axillary six, and in the inter-scapular line three inches.

Spleen.—The spleen lies in the left hypochondriac region, with its long axis parallel to the ribs. It is a flattened oval measuring five by three inches. Its landmarks ought to be thoroughly understood, for its relationship to the stomach, colon, and kidney is in certain abnormal conditions of great importance. The tenth rib may be taken as corresponding to its position. Its posterior pole is found about one and a half inches from the middle line at the tenth dorsal spine, and its anterior pole comes round about as far as the mid-axillary line—sometimes as far as the anterior axillary line. The upper third, posteriorly, lies under the diaphragm, and is overlapped by lung tissue, while the lower two-thirds anteriorly lie in contact with the parieties. The left kidney lies behind the spleen. The method of examination, to delimit the spleen, is ery similar to that which is employed for examination of the kidney. Except when greatly enlarged, and projecting well into the abdomen, when it can be seen freely moving during respiration, inspection should reveal nothing.

To carry out palpation to most advantage, and to attain the greatest degree of relaxation, the dorsal decubitus should be adopted with the head and shoulders on a high pillow, the knees well flexed, and the right arm in extended position pulled over across the chest till the hand lies in the left groin.

In the first instance palpation with one hand ought to be carried out in the ordinary way. The right hand is placed flat on the abdomen, with the fingers pointing towards the ribs in the hypochondriac region. The finger tips are gradually inserted upwards under the costal arch and palpation made with their pulps during

quiet and forced respiration. If nothing be felt, there is no enlargement of the spleen. A spleen palpable by this method is to be suspected of slight enlargement. The fingers ought to search for the sharp and firm edge of the organ and its characteristic and diagnostic notch.

A better examination of the enlarged spleen can be made by the bi-manual method. With the physician standing on the right side and bending over the patient, the left hand should be inserted under the ends of the tenth and eleventh ribs, and a firm lifting pressure exerted upwards. The fingers of the right hand, insinuated under the margin of the ribs in front, will compress the organ between these and the underlying left hand. It can be felt both behind and in front, and its free movability during respiration made out. Its sharp edge and its characteristic notch, or notches, readily differentiate it from other organs.

Percussion is not nearly of such diagnostic value as palpation, because the spleen is overlapped by lung posteriorly. Its absolute, or superficial, dulness can be best mapped out by gentle percussion. The patient should lie in the recumbent position on the right side, with the body turned slightly over towards the ventral decubitus. If dulness can be made out beyond the mid-axillary line it indicates enlargement.

In certain morbid conditions the size of the spleen is variable from time to time, as, for example, in malaria and septicæmia. It should also be noticed that the spleen may be displaced by disease in neighbouring organs. Thus it is depressed in left pleural effusions and is sometimes erroneously diagnosed as enlarged. It may also be floating, forming part of the picture of Glénard's disease.

In meteoric distension of the abdomen the spleen cannot be percussed at all with any precision. A loaded bowel or full stomach may give rise to a dulness, which blends with and masks the true splenic dulness.

The area which normally lies between the anterior border of the spleen and the left border of the liver is tympanitic. It is known as *Traube's space*, and is of importance because in pleuritic effusions splenic dulness cannot be made out owing to the obliteration of this tympanitic area. The spleen must always be differentiated from the left kidney.

Kidney.—Certain abnormalities of the kidney may give rise to a group of symptoms not dissimilar to those which may occur in

gastro-intestinal derangements. No examination, therefore, of a patient suffering from dyspeptic symptoms would be complete without a careful physical examination of these organs. So many symptoms have been assigned, usually quite wrongly, to a movable or floating kidney that this condition must be considered in some detail.

The normal kidney cannot be mapped out with any precision by any of the usual methods of physical examination. The upper pole of the right kidney lies in contact with the liver—and the left with the spleen. The kidneys are retro-peritoneal, and are maintained in their positions chiefly by a pad of fat and connective tissue. The right kidney lies about a half to three-quarters of an inch lower than the left. When the abdominal wall is thin and relaxed it is possible to employ palpation with a considerable degree of accuracy. The patient should be in the dorsal decubitus, and the same measures adopted to attain relaxation of the wall as for the spleen (q.v.) The kidney should be searched for on each side by the palpating hand pressed deeply under the costal arch in front and obliquely backwards towards the spine. They should be examined during both quiet and forced respiration. The bi-manual method of examination is very useful when the organs are enlarged or displaced. The edge of the lower border of the right normal kidney can often be felt by the palpating hand when the abdominal wall is thin and well relaxed ; but if more than the edge can be felt, it is certain that the organ has suffered some downward displacement or is enlarged.

Movable Kidney.—Movable kidney must always be sought for in any abdominal examination. I am convinced that far too many symptoms of ill-health and abdominal derangements are ascribed to ' movable ' and ' floating ' kidney. Any one who has many patients passing through his hands, in the wards of a hospital, can easily verify for himself that not less than half of the female inmates has ' palpable,' ' movable,' or ' floating ' kidney. Many writers give even a higher percentage than this. In describing a condition of undue movability of the kidney it must be remembered that different examiners have a different standard for this condition. The great majority of patients who are subjects of this abnormality have no knowledge of its existence and suffer no symptoms from the condition. It should be observed that all degrees of displacement may take place. The slightest movability

to the most extreme dislocation may be found. Those kidneys that merely are easily palpable, but which remain in the hypochondriac region, and can by pressure be made to slip up and down in their long axis, but practically in their normal position, may be described as ' movable.' On the other hand those which have left the neighbourhood of their normal locality are spoken of as ' floating.' There is no advantage in classifying these displacements in exact correspondence to their degree.

Undoubtedly the best method of examining, for the movability of a kidney, is to get the patient out of bed, make her rise on the toes, and suddenly and forcibly fall on the heels two or three times in succession. If such a manœuvre, coupled with a few forcible coughs, fails to bring a kidney downwards from its normal position, it is presumably anchored in the loin in its natural situation. While the patient is still erect, the examiner should firmly grasp the flank, with the left hand for the right kidney and with the right hand for the left kidney, the thumb pressing anteriorly deeply under the costal arch and the fingers behind. By this procedure the organ, if it has fallen downwards, is retained in its abnormal position, with the thumb and fingers above its upper pole. While still grasping the flank in this manner, the patient is slowly lowered to the recumbent dorsal decubitus, and the right hand placed first flat in the sub-costal region, and the kidney felt for. If it cannot thus be palpated, the pulps of the fingers should then be pressed inwards and upwards, and the organ will, if displaced, be readily felt. The contour, consistence, and tenderness of the organ will aid the diagnosis. When such a kidney is felt, a continued firm pressure with the right hand, while the left hand still compresses the tissues in the flank above the kidney, will often result in its suddenly slipping up between the compressing thumb and fingers, with a smooth gliding motion which is absolutely characteristic.

A ' floating ' kidney may be found anywhere in the abdomen, and may pass well below the umbilical region, or even as low as the groin.

The right kidney is far more commonly displaced than the left. Probably the pressure of pregnancies, coupled to the close relationship which the right kidney has to the large solid liver, which acts as a fulcrum, is responsible for this.

Differential Diagnosis of the Kidney and Spleen.—When palpating the left hypochondrium the examiner may find a tumour in

that region, and may be in doubt as to its nature. Is it spleen or kidney ?

The spleen should be differentiated from the kidney by these points. It lies more superficially, and there is no part of the hollow viscera between it and the abdominal wall, with which it is in close contact. The colon passes between the kidney and the abdominal wall, hence, especially if the colon be inflated, an area of tympanicity, traversing the dulness, can readily be made out. No such break in the continuity of the dulness is found in splenic enlargements or displacements. The splenic notch, if it can be found, is absolutely characteristic. The spleen is much more movable during respiration than the kidney.

The Etiological Factors in Movable Kidney are diverse. The condition is rarely traumatic. It is far more commonly associated with a congenital weakness of the tissues. It is sometimes said to be acquired, but in reality the inherited predisposition is the chief factor in the causation of the abnormality.

Movable or floating kidneys are found in conjunction with a long mesonephron and with a general relaxation of the abdominal wall and undue movability of other organs (see Gastroptosis).

In those who have inherited the predisposition many factors may contribute to renal displacements. Over-frequent pregnancies, tight-lacing, and strain are all credited with the production of this abnormality. The greater degrees of ' floating ' kidney are found in association with Glénard's disease. Some cases of general enteroptosis give rise to no symptoms at all, while others are associated with pronounced local and general disturbance, usually of the neurasthenic type. The ' papery ' abdominal wall, with its emaciated and gaping recti, reveals the distinct outline of the underlying viscera. In such conditions visible normal peristalsis of the stomach and bowel can often be seen. The condition may be associated with atonic gastric dilatation with, or without, alterations in the secretory and motor functions of the stomach. The kidneys, spleen, liver, and colon are often greatly displaced in this condition, and the transverse colon may bend downwards so as to form a V-shaped displacement with its apex well below the umbilicus.

Symptoms when they occur are usually referred to the abdominal cavity or reflexly to remote parts. Pain and dragging, in the lumbar region, many and various dyspeptic symptoms and those

of neurasthenia, are frequently associated with Glénard's disease. Displacement of the kidney alone rarely gives rise to any symptoms of its presence, and the condition is discovered, as a rule accidentally, during abdominal palpation. I have found that it has been principally in those subjects who *know* they have a floating kidney that symptoms are complained of at all. While this is true of the majority of such cases, undoubtedly a floating kidney may at times apparently give rise to many and various abdominal symptoms and to considerable reflex disturbance, even when the patient has no knowledge of the existence of the displacement. The serious mental disturbances sometimes assigned to this trouble must be, indeed, of rare occurrence.

If a displaced kidney is unduly tender to palpation, and shows any signs of enlargement, a careful examination of the urine should be invariably made. The presence of any appreciable quantity of albumen or any trace of blood is indicative of some hyperæmia, probably due to kinking of the vessels of the hilus.

Perhaps one of the most important accompaniments of movable kidney is that of gastric dilatation, probably brought about by pressure exerted on the duodenum. Some surgeons insist that it occasionally may cause pressure on the bile ducts, and so produce jaundice. This, too, must be a rare condition. The general enteroptosis described by Glénard, which is found in certain cases of movable kidney, is the result of a general relaxation of the abdominal wall and intra-abdominal tissues and loss of supporting fat.

From time to time an urgent condition may arise in those suffering from movable kidney, and symptoms supervene which are not infrequently mistaken for a gastro-intestinal, renal, or biliary colic. The sudden onset of severe abdominal pain accompanied by nausea, vomiting, fever, and collapse in cases known to have a floating kidney, should always suggest the possibility of a *Dietl's crisis*. A careful abdominal and urinary examination, with an accurate clinical history, may enable the diagnosis to be made. This condition may be brought about by kinking of the ureter and vessels, or by an aberrant artery passing under the ureter, and the over-distended pelvis of the kidney may be felt as an abdominal tumour. Sometimes kinking may result in the retention of urine in the kidney, without the symptoms of a Dietl's crisis, and produce a passing hydronephrosis.

Treatment of these conditions is not always easy. If a kidney is accidentally found to be displaced, it is probably unwise to inform the patient. The knowledge of the existence of this abnormality is a fruitful source of hypochondria. Too often such cases are operated on. Fixation of the kidney is sometimes, but not by any means always, successful in relieving the symptoms. It may have a ' moral ' influence on the nervous patient. As the kidney is largely maintained in its position by a pad of fat, and its displacement occurs principally in thin subjects. a line of treatment suggests itself. A thoroughly carried out ' rest-cure,' with massage and regulation of the diet and bowels, may result in a rapid gain of weight and new fat formation which may anchor the kidney in its normal position or diminish its movability. Pads and belts, which aim at retaining the kindey in its normal situation, I have not found very useful.

When general enteroptosis exists, abdominal support is usually more successful. A well-made pair of straight-fronted abdominal corsets may exert a sufficient amount of pressure to compensate for the undue relaxation of the abdominal walls. Many belts of different patterns are recommended by various writers, but anyone which gives comfort, combined with good support, will best answer the purpose.

The Pancreas.—The intimate relationship which exists between intestinal and gastric digestion makes a study of the pancreas essential. The organ is not, in its normal condition, accessible to physical examination. It lies about half way between the ensiform cartilage and umbilicus, with its long axis transversely across the abdomen at about the level of the second lumbar vertebra. Its length is about six inches. It has very important anatomical relationships to other structures, which are not infrequently involved in its disease. Behind the head of the pancreas lie the inferior vena cava, portal vein, and the common bile duct. In about 70 per cent., in the human subject, the bile duct passes through the head of the pancreas.

Tumours of the head of the pancreas, when sufficiently large, may press on all these structures and give rise to the evidences of portal obstruction with jaundice or ascites from pressure on the venous return ; while pressure on the solar plexus may give rise to polyuria or glycosuria.

In front of the pancreas lies the stomach. Ulcers on the posterior wall of the stomach not infrequently become adherent to the pancreas and may deeply erode its tissue. Such cases are usually associated with severe and persistent pain in the back.

Tumours of the pancreas may involve the gastric wall to which they become adherent. Such growths tend to bulge into the epigastric region. They are fixed, and do not move on respiration. We are, owing to the deep position of the pancreas, dependent more on certain symptoms and signs, than on any physical change, for the diagnosis of diseases of this gland.

CHAPTER VIII.

EXAMINATION OF THE STOMACH BY CLINICAL METHODS.

Physical Diagnosis.—The stomach is best examined in the dorsal decubitus. It is, however, often necessary to examine the viscus when the patient lies on one or other side, and occasionally in the knee-elbow position as well as when the patient is erect. The abdominal wall should be as much relaxed as possible. If this cannot be attained by flexion of the knees, an anæsthetic may be required. The stomach should be examined both when full and empty, and during ordinary and forced respiration.

(1) *Inspection.*—This method may often yield important information in the presence of gross abnormality, but little can be made out when there is no pronounced change present.

Usually, in the subject who is not obese, the gastric region is flat or slightly hollowed when the stomach is normal. But it may present a distinct fullness or bulging when the organ is distended. Its outline can sometimes be studied when it is inflated.

Visible Peristalsis can be seen in many cases where there is stenosis at the outlet. The waves of contraction usually pass from left to right, but not infrequently there are anti-peristaltic waves running in the opposite direction. Peristalsis in the stomach can sometimes be evoked or stimulated by scratching the flanks in the region of the ninth to the twelfth ribs, while flicking the epigastric region may also cause contractions. Kussmaul has noted that in pyloric stenosis, with dilated stomach, there may be " peristaltic restlessness " with waves passing from the linea alba below the level of the umbilicus in an upward direction towards the right costal margin.

Often local bulgings from tumours can be seen in thin subjects, and on deep inspiration it may be noted that they have considerable freedom of movement.

When the patient is inspected, in a good light, in the erect position, in gastroptosis, the epigastric region is often scaphoid and the umbilical region full from the displaced and often distended stomach.

(2) *Palpation.*—This is a more informing method than that of inspection. It should be carried out, for the stomach, as indicated in the section on abdominal examination (*q.v.*).

With the legs flexed, and the mouth open, the necessary degree of relaxation is usually attained.

Often peristalsis, noted by inspection, can be felt.

Tumours can be located if they are not situated too high up in the cardiac end. By deep inspiration these are sometimes brought partially to the palpating hand. When inflation, by means of the stomach tube and a Higginson's syringe, is carried out simultaneously, one can determine, often with considerable accuracy, whether a tumour is situated on the posterior or anterior wall of the stomach. (See inflation of stomach, page 88).

All alterations in the resistance of the abdominal wall, wherever this condition may be present, must be carefully noted. The upper bellies of the recti should always be carefully examined for any increased rigidity.

Any tender or hyperæsthetic spots must also be looked for.

Splashing sounds are well elicited by sudden dipping palpation, but often better by grasping the patient with the hands placed round either flank and shaking him from side to side. The amount of fluid present in the stomach can be well estimated by the amount and size of impact felt by the hand, as the fluid ' ballottes ' from one side to the other.

One should always attempt to delimit the lower border of the stomach by means of dipping palpation, and noting the lowest point at which splashing sounds are heard. Succussion, at the lower border of the stomach, is best evoked by palpating from below upwards—starting from a point well below the umbilicus. If we are in doubt as to the origin of a succussion or splashing sound, produced by this thrusting palpation, in the epigastric region, the stomach contents should be removed by the tube and extractor, and if the sound still persists it is clear that it is due to colon. It should be remembered, in this connection, that the colon contents are fluid as far as the splenic flexure.

Succussion is not always an abnormal sign. It is really only

pathologic when it occurs at longer intervals after food than it ought to. Ideally the stomach ought to clamp its contents, and have a sufficient tonicity to evacuate any gas which may be present. It would then behave as a bottle filled with fluid to the very cork, when no amount of shaking could elicit splashing. These conditions are rarely existent even in people who have a sound digestive apparatus. Gastric succussion implies the presence of fluid and air and a not too great tonicity of the gastric wall. Even when very abnormal splashing, far too low to be within the range of normality, is elicited with ease in the dorsal decubitus, it becomes often quite impossible to evoke it when the patient is in the perpendicular position, because the large volume of fluid present gathers in the most dependent part, and, by dragging on the stomach wall, forces it to clamp its contents firmly while the air passes into the cardia below the diaphragm. Under certain conditions gastric succussion has great diagnostic value, and is typical of dilatation. After a mixed meal, splashing can be detected in many healthy people for a considerable time. Splashing or *clapotage*, which is produced by the smallest shake or by the lightest tapping palpation, is always abnormal if it be too low down, and can be demonstrated to have a gastric origin. Splashing confined to the upper half of the epigastric and hypochondriac regions is more or less normal in most people. As fluid in the normal stomach rapidly leaves the viscus, it follows that succussion habitually detected at long intervals after ingestion is abnormal, and indicates a ' motor insufficiency.'

It should be remembered that the conditions which make clapotage possible are found in large subphrenic abscesses, and that splashing ought to be sought for, by briskly moving the patient from side to side, in those cases where this condition is suspected. Generally speaking gastric splashing, which can be elicited below the umbilicus, indicates abnormal relaxation of the stomach walls, and probably some ptosis of the organ.

(3) *Percussion of the Stomach.*—Percussion alone yields unsatisfactory results, because so much depends on the condition of the colon and other air-filled organs. If the colon and small gut be distended with air to an equal extent to the stomach, the note may vary so little that no precise information can be gathered.

The stomach, in its natural condition, gives a lower-pitched note than the small intestine, but the exact amplitude and pitch

of the percussion note will greatly vary, with the amount of distension present, in the colon and small intestine. It is clear that if the stomach be completely collapsed and contains no air percussion will give no information. The tympanitic note, heard in the upper epigastric and left hypochondriac regions, is due rather to the colon than to the stomach, when the latter organ is empty.

The upper border of stomach note can usually be made out, with some degree of accuracy, but the lower border is more difficult to locate, because the lower curvature readily falls away from the abdominal wall. When the stomach is very full, and gas is absent, the note is absolutely dull. It should be remembered also that Traube's space is obliterated in large left pleural effusions.

(4) *Auscultation of Stomach* is of some value in the physical examination of the viscus. Obstruction at the cardiac end of the œsophagus can sometimes be detected by this method.

When the stomach is atonic, and contains a considerable quantity of fluid and air, succussion may be produced by the cardiac systole, the beat of the aorta, or by the descent of the diaphragm during respiration.

When solutions of sodium bi-carbonate and tartaric acid (one drachm in each) areintroduced into the stomach to produce inflation the sizzling sound produced by the liberation of bells of gas, during effervescence, can be readily heard through the stethoscope. By this manœuvre the lower border of the stomach can best be located if the procedure be carried out when the patient is standing.

(5) *Auscultatory Percussion* is a more valuable method of delimitating the position of the normal and abnormal stomach. Although many observers maintain that it is not reliable, I have found that if this method of physical diagnosis is carefully carried out, the findings correspond sufficiently accurately to those obtained by opaque meal and X-rays. The rationale of this method depends on the different notes produced by the different organs according to their densities.

There are many ways by which this procedure can be carried out. Either percussion, scratching, or flicking may be employed. A binaural stethoscope, with phonendoscope ending, gives more emphatic results. The dorsal decubitus is the best position for this examination, and a plesser and pleximeter are sometimes useful. It is employed to determine the size, position, and outline of the stomach. Even at the part overlapped by solid liver, the

clear percussion note is transmitted through the solid organ. The results are, of course, not exact, but only approximate. The chief source of error is from a distended colon, which masks the true lower border of the stomach.

The technique is as follows :—

(*a*) The mouthpiece of the stethoscope is placed over the normal position of the fundus—that is, about the sixth or seventh rib in the mammary line.

(*b*) Percuss, or scratch with a spicule of wood (an ordinary wooden match somewhat sharpened answers well), *close to* and around the stethoscope, so as to familiarise the ear with the character of the true stomach note.

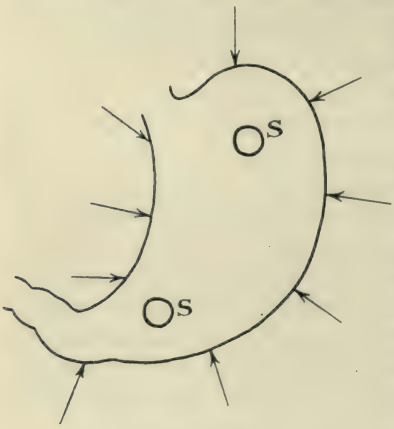


Fig. 15.

Diagram illustrating Methods of Auscultatory Percussion of Stomach.

S. Position of stethoscope.
→ Line of percussion or scratching.

(*c*) Then begin percussing, or scratching, at several points obviously distant from the stomach—from above downwards, from below upwards, and from both sides towards the stethoscope. Whenever the border of the air-filled stomach is reached, the note conveyed to the ear will become clearer and of higher pitch. Each point at which the characteristic stomach note is reached should be marked by a dot with the skin pencil, and, at the end of the examination, each dot joined up so as to figure the outline.

If it be found, as it may be, that the maximum note is not at the normal fundus position, the stethoscope should be moved till the clearest and highest note be determined. If the stomach be empty and collapsed, auscultatory percussion is useless. It is, therefore, essential for accuracy, to aspirate any contents, and before removing the syphon to inflate the organ to a moderate extent by means of a Higginson's syringe.

Auscultatory percussion is of value, after some experience has been gained, in determining the situation of a solid tumour in the

stomach region and in differentiating whether it takes origin in the stomach or in the liver. This method can be employed to differentiate growths in the liver or gall-bladder from those of the stomach itself.

Examination of the Stomach by means of Inflation.—This procedure is contra-indicated after recent hæmorrhage, or where ulceration with pain (indicating perigastric inflammation) is present.

This method gives most valuable information, for in all cases where the abdominal wall is not too thick or rigid the actual outline of the stomach can be seen and felt at a certain stage of inflation through the abdominal wall.

But in addition it is not a bad test of the tonicity of the gastric wall. When the stomach is healthy, and its musculature normal, it resents any great distension, and contracts on its contents, expelling the air between the tube and the œsophageal wall. The atonic stomach can be inflated almost to any extent, without discomfort to the patient, and without the expulsion of the gas. Any considerable distension of the healthy stomach is at once resented, and produces considerable pain. Besides affording such information on the above points, the position of a tumour, taking origin in the stomach, can often be accurately determined to be attached to the posterior or anterior wall. In the former case inflation makes the tumour recede; in the latter it projects it more on to the abdominal wall, when it can either be seen or felt.

When the stomach is inflated, both ordinary and auscultatory percussion become more easy and reliable.

There are two methods of causing gaseous distension of the stomach :—

(1) *By Carbonic Acid* generated from the administration of two solutions (each of about six ounces of water) containing one drachm of sodium bi-carbonate and tartaric acid. If the stomach is known to be large, a drachm and a half of the soda and of the acid may be given. The soda solution should be swallowed first. I do not care for this method, as being less accurate than the second, described below. Sometimes the gas, owing to too rapid formation, is belched up or escapes by the pylorus. But a more serious objection is that it occasionally causes, from over-distension, great pain, and it may induce a serious syncope, as I have seen on at least one occasion.

(2) *Inflation with air.*—For this purpose an ordinary sized stomach tube and a Higginson's syringe are required. This method, by the ease with which the amount of air introduced can be regulated, avoids all the inconveniences and dangers of the carbonic acid inflation. The conditions which contra-indicate the introduction of the tube and the technique to be observed are discussed in the section on the examination of the stomach by means of the tube. Before air is introduced the gastric contents should be extracted. During the process of inflation the patient should lie in the dorsal position, with the knees drawn up. When sufficient distension has been produced to demarcate the outline of the stomach, the position of the lower border (greater curvature) should be outlined by the skin pencil. In gastroptosis often the smaller curvature can be easily demonstrated by a moderate amount of inflation. Inflation will often evoke peristaltic movements that had not been previously noted, and demonstrate an obstruction at the pylorus.

When the examination is over, before the tube is removed, the gas should be allowed to escape through it. Nearly the whole of it can be expelled by pressure, with the flat of the hand, on the epigastric region.

(3) *Distension by means of Water* (Dehio's method).—This procedure consists in percussing the stomach in the erect position after it has been completely emptied of its contents. Note its lower border. Then give successive glasses of water, each containing eight ounces, and after each glass note the position and outline of the stomach. If the stomach be over-distensile and atonic it will be found that, in the erect position, the area of dulness lowers and broadens after each glass. In extreme cases of dilatation and ptosis the stomach dulness will be found to descend even down to the pubis. The amount of atony may be roughly estimated by the water test with an accuracy nearly equal to that of inflation. When the patient is placed in the recumbent position the area of dulness will have disappeared, which demonstrates that it was due to the introduced fluid. In many instances, where great atony has existed, I have noted that the left half of the abdomen, with patient in the erect position, has been bulged outwards by the distended organ.

(4) *Other Methods.*—In addition to the foregoing the following methods of investigation of the position and size of the stomach

may be mentioned. But some of them are not so safe or so valuable.

The introduction of a solid bougie, as far as the greater curvature, and by palpation, the determination of its situation through the abdominal wall is not a method that in all cases would be safe.

Gastroscopy.—This method, based on the direct observation of the appearances of the œsophageal and gastric mucosa, requires much technical skill, and subjects the patient to great discomfort and to some danger. An anæsthetic is frequently required to carry it out. As far as I have seen its use, I am not convinced that the information to be derived from it is superior to that to be gained by other, and less troublesome, methods of observation.

Transillumination of the empty stomach, by the introduction of an electric light after Einhorn's method, may be employed. It will roughly outline the stomach, and may show any tumour on the anterior wall. The procedure is resented by the patient, because the tube employed requires to be large, but undoubtedly information as to the size of the stomach and the presence of a tumour may be obtained with a considerable degree of accuracy by this method.

X-rays in Gastric Diagnosis.—In expert hands, and with good apparatus, the examination of the digestive tract by means of bismuth or barium meals and X-rays, forms one of the most promising modes of investigation.

Raying may be employed for a great many purposes, as for the detection of metallic foreign bodies in the stomach or intestine, for ascertaining the size, position, and motility of the stomach, and for detecting the position of any obstruction in any part of the tract.

CHAPTER IX.

RADIOGRAPHY.

There can be little doubt that recent advances in gastro-intestinal diagnosis have been largely dependent on Radiography.

In these days, when clinical methods run the risk of being eclipsed by laboratory investigations, the diagnostic value of X-rays in elucidating gastro-intestinal diseases may well be overestimated. There can be no doubt, however, that we owe a great deal to radiographic investigation for locating certain lesions and indicating their nature. This is specially the case in the domain of surgery.

Just as it is impossible to get complete diagnostic data from the clinical history and a study of the gastro-intestinal chemistry, so it is also expecting too much to seek from a radiographic investigation certain demonstration of the nature of the disease.

So far as the stomach is concerned, other physical methods of examination are not inferior. Soundage, inflation and gastric diaphany may give information as valuable as that obtained by radiography. The impediments to the more universal employment of X-ray examination are the expense and the difficulty of getting patients, who live in places remote from cities, to submit themselves to the investigation.

If X-ray work is to be of any value it must be carried out by an experienced radiologist in possession of adequate apparatus.

Preparation of the Patient.—A little simple preparation is necessary. The bowels should be well moved two days before the opaque meal is to be taken. The exaggerated peristalsis induced by the purgative may still persist if the aperient is taken the day previous to the investigation and give an erroneous impression of the motor function of the intestine.

It is not sufficient to merely get a good negative. The physician ought to be educated to interpret correctly the appearances of the radiogram. This takes some time and training, and for those unaccustomed to study radiograms the interpretation of the radiologist ought to be accepted.

Four ounces of barium sulphate is now given in preference to bismuth carbonate. It is cheaper, and less likely to contain toxic matter. The opaque salt should be mixed with gruel, or given in buttermilk. The mixture is often very distasteful to certain patients. It is less unpalatable if administered in a pint of sweetened and flavoured arrowroot gruel of a thick creamy consistence.

The position of the opaque meal should be noted immediately after its entrance into the organ which is being examined, and its position thereafter noted at fixed intervals of time.

It is essential to know the normal time taken for the stomach to empty itself and to remember where in the small and large intestine the opaque meal ought to be found after the lapse of definite periods of time.

The normal stomach begins to empty itself at once of the barium meal. The rate depends on the motor function and on the patency of the pylorus. If there be a delay in emptying beyond four hours, we may conclude that there is either loss of motility or interference with the outlet.

Once the opaque meal leaves the stomach, it very quickly passes through the normal small intestine and the head of the meal reaches the colon in from three to six hours. Usually it arrives at the splenic flexure in about ten hours after ingestion.

From these normal times there may be every degree of variation and delay, from some hours to days, according to the lesion present.

Normally the colon can be examined in twenty-four hours after the opaque meal has been taken, but it may be necessary to administer the barium by enema when a thorough examination of the lower gut has to be made.

Radiographic investigation is suitable for elucidating a large number of diseases. The œsophagus, stomach, intestines, biliary and renal apparatus may be so examined.

(1) Œsophagus
- Diverticula.
- Certain foreign bodies—*e.g.*, coins, bones.
- Stenosis
  - Spasmodic.
  - Organic—*e.g.*, tumours, cicatrices.

- (2) Stomach
  - Form, size, and shape
    - Dilatations.
    - Gastroptosis.
    - Deformities (hour-glass, adhesions).
    - Tumours.
    - Ulcers.
    - Foreign bodies.
  - Motor functions
    - Hypomotility.
    - Hypermotility.
    - Atony.
    - Stagnation.
    - Pylorospasm.
    - Pyloric Stenosis.
- (3) Intestines
  - Foreign Bodies.
  - Displacements and Deformities.
  - Motor functions.
  - Obstructions.
- (4) Urinary Apparatus
  - Size of Kidney.
  - Renal Calculus.
  - Calculus of Ureter.
  - Vesical Calculus.

(5) Biliary Apparatus.—Gall stones in bladder and ducts may be observed.

The Œsophagus.—If the opaque meal can be got to enter a *diverticulum* it will show its position and size. A thick tenacious emulsion of barium is best for examination of the gullet, as it tends to pass less quickly into the stomach and gives a better shadow. For the demonstration of obstructions radiography is useful. A capsule of barium often answers better than the emulsion as it remains above the sight of the stenosis till it melts or is regurgitated. Objection may be taken to its use, as it at times produces spasm.

The Stomach.—The appearance of the stomach—size, outline, and form—should be studied. One of the most important points to observe carefully is the pylorus. Its form, its movements, and its capacity to permit the passage of the opaque meal should be

noted. These observations can all be made on the fluorescent screen.

In all cases a radiogram should be taken of the stomach in the erect and recumbent postures. The position of the stomach in these two positions differs greatly, and in certain diseases it is the position of the organ in the erect which is of vital importance in the diagnosis. We might well fail to detect a considerable degree of ptosis or dilatation if we had to depend solely on a radiogram taken in the recumbent posture.

It should be kept in mind that a stomach filled with an opaque meal is bound to be pulled somewhat downwards by the weight of its contents. Due allowance must be made for this in estimating the position of the lower pole of the stomach.

In the recumbent position the lower pole of the empty or moderately filled healthy stomach extends down to about one inch above the umbilicus, while in the erect posture it usually comes down one inch or more below it.* The pyloric portion passes by 1 to 1½ inches to the right of the middle line.

The upper two-thirds of the normal stomach are vertical, and the lower one-third is nearly horizontal; but the exact shape and position of the normal stomach are subject to great variation.

The Duodenal Cap.—This shadow is of great importance. In the normal stomach it is situated above the pylorus but separated from it by a defined clear space which represents the position of the sphincter. In shape it is like a small bee-hive, and represents the opaque meal in the first part of the duodenum (see diagram of radiogram). Certain alterations in size, shape, and density of the cap may be regarded as significant of special lesions and conditions.

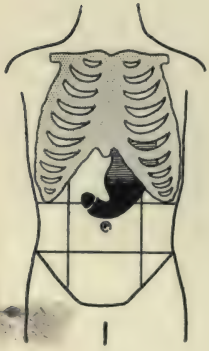
In pronounced pyloric obstruction there is a complete absence of duodenal cap.

If the stenosis be incomplete and the opaque meal can trickle through, the cap is displaced, deformed, and incomplete.

The duodenal cap is also ill-defined in pronounced gastric atony, while duodenal ulcer may be suspected when the cap is in normal position but is irregular in its outline.

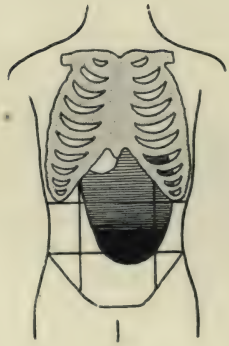
When gastric peristalsis is very active, the waves of contraction may so deeply indent the borders of the organ that the appearance

* The umbilicus, like the nipple, is not a good landmark owing to its variable position. We may with advantage substitute for it the intercristal line.



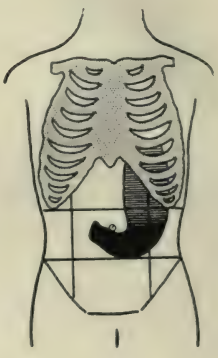
1.

NORMAL STOMACH



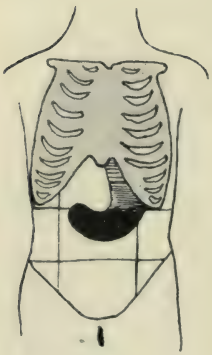
2.

ATONIC DILATATION.



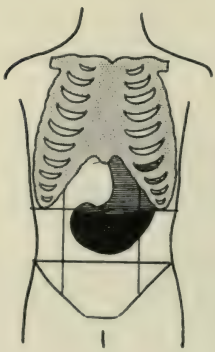
3.

OBSTRUCTIVE DILATATION.



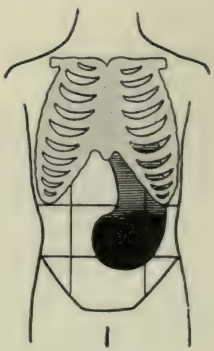
4.

MINOR GASTROPTOSIS



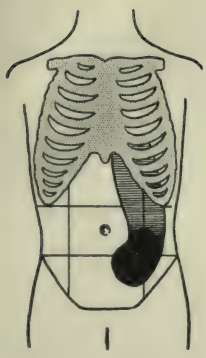
5.

MODERATE GASTROPTOSIS.



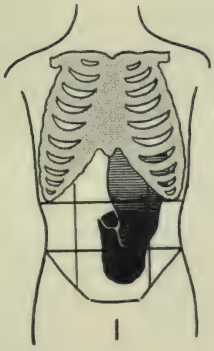
6.

STOMACH BECOMING VERTICAL.



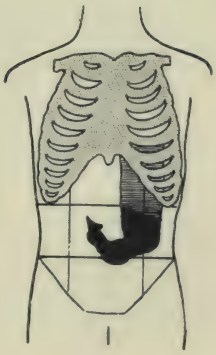
7.

More Advanced Vertical Stomach.



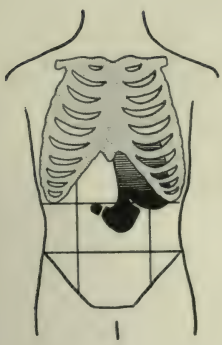
8.

Water-trap Stomach.



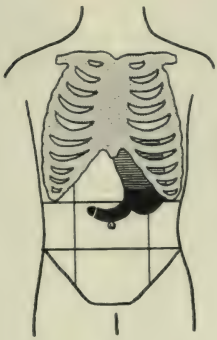
9.

Fish Hook Stomach.



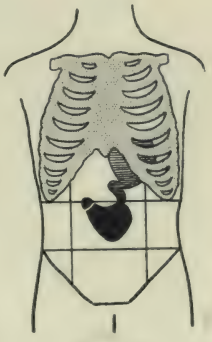
10.

Hour-glass Stomach.



11.

Hour-glass Stomach.



12.

Hour-glass Stomach.

of a double sac may result. Hour-glass contraction has thus erroneously been diagnosed by those inexperienced in reading skiagrams.

Again, at the site of a gastric ulcer, the irritation produced by the lesion may result in a violent local spasm which gives rise to an appearance differing little from that of a true hour-glass deformity. Care should be taken to exclude spasm before deciding that organic deformity exists.

Manipulation of the stomach, while the opaque meal is being screened, may quickly dissipate the shadow or demonstrate that it is not a permanent abnormality and, if the clinical symptoms and signs are not those that are typical of hour-glass contraction, a dose of atropine, administered before the investigation, will also have the effect of removing the local spasm. If neither manipulation nor the action of a full dose of atropine results in altering the indentation, we may conclude that it is not spasmodic but organic.

The diagrams of skiagrams following illustrate some of the common abnormalities :—

Dilatation of the Stomach.—The radiograms have different appearances, according to the degree of dilatation and its cause.

(*a*) The atonic type of dilatation shows that the lower pole of the stomach is sagged downwards. The stomach as a whole may assume various shapes, and the lower pole may be found in any position between the umbilicus and the symphysis pubic.

The tonus and the peristalsis are practically absent, and usually the duodenal cap is absent or defective.

(*b*) Obstructive dilatation.—The nature of the skiagram depends on the condition of the gastric muscle. In very chronic cases, when compensation has failed, the type of radiogram may be that of the atonic stomach. But as long as compensation is fairly well maintained, the lower pole of the stomach is not so low (although it may be more horizontal) as that found in atonic dilatation.

Gastroptosis.—In this condition the stomach varies greatly in form and position. Gastroptosis is not infrequently complicated by dilatation, and we find all degrees of displacement as the disease progresses.

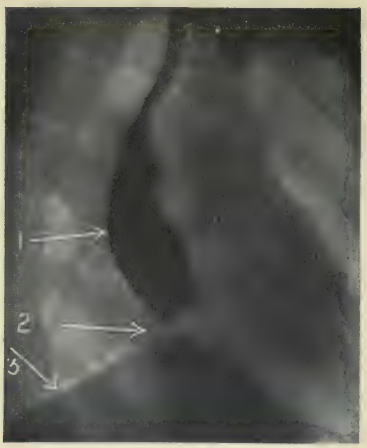
The essential feature of gastroptosis is the descent of the smaller curvature. In those cases of very long standing the stomach

(1)

NORMAL STOMACH.

(1) Peristaltic Waves.
(2) Pylorus.
(3) First Part of Duodenum.

(2)



OBSTRUCTION OF GULLET LOW DOWN.

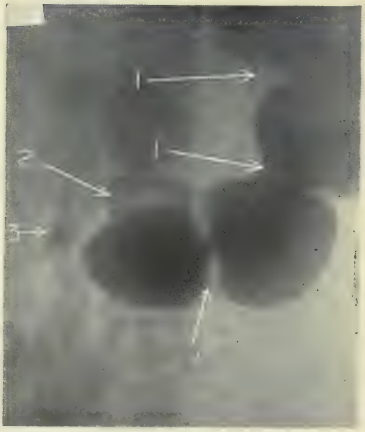
(1) Dilated Gullet above Stricture.
(2) Site of Stricture.
(3) Diaphragm.

(3)

OBSTRUCTION OF GULLET BY A POUCH.

(1) Pouch.
(2) Narrow Gullet.

(4)



HYPERPERISTALSIS DUE TO PYLORIC OBSTRUCTION.

(1) Peristaltic Waves.
(2) Narrowed Pylorus.
(3) Duodenum.

PLATE III

(5)

NORMAL STOMACH WITH SPASM AT PYLORUS.

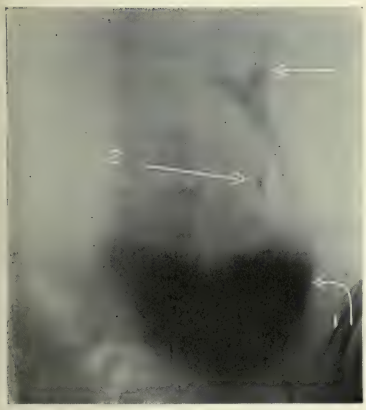
(1) Site of Pylorus.
(2) Peristaltic Wave.

(6)

CHRONIC ULCER ON LESSER CURVATURE.

(1) Crater of Ulcer.

(7)



ATONIC DILATED STOMACH WITH CHRONIC ULCER ON LESSER CURVATURE.

(1) Stomach.
(2) Crater of Ulcer.

(8)

ULCER WITH ATONIC DILATATION OF STOMACH.

(1) Crater of Ulcer at beginning of Duodenum.
(2) Duodenum.
(3) Dilated Atonic Stomach.

PLATE IV

(9)

CHRONIC ULCER ON LESSER CURVATURE CAUSING HOURGLASS CONTRACTION AND PYLORIC STENOSIS.

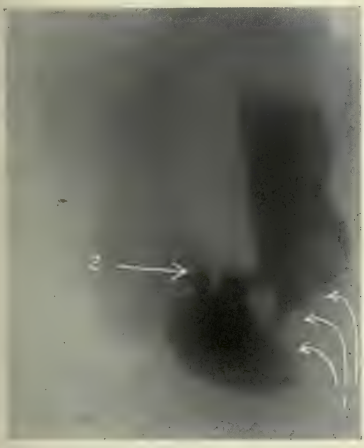
(1) Walls of Ulcer.
(2) Crater of Ulcer.
(3) Hourglass Contraction.
(4) Portion of Stomach distal to Contraction.

(10)

CHRONIC ULCER AND HOURGLASS CONTRACTION.

(1) Diaphragm.
(2) Crater of Chronic Ulcer.
(3) Contracture forming Hourglass Stomach.
(4) Operation Stoma.
(5) Small Bowel.

(11)



TUMOUR OF GREATER CURVATURE.

(1) Large irregular "Shadow Defect" caused by the Tumour.
(2) Pylorus.

(12)

TUMOUR OF STOMACH.

(1) Extensive "Shadow Defect."
(2) Pylorus.
(3) Duodenum.

becomes more and more vertical. The pylorus remains fairly fixed, so that while the cardiac end droops the pyloric portion remains comparatively high, giving rise to the 'fish-hook' and 'water-trap' stomachs. (See various diagrams of Gastroptosis.)

Hour-glass Stomach.—This deformity presents many variations. The seat and extent of the ulcer or tumour, and the subsequent cicatrization, determine the position of the contraction. It is, however, most commonly found at the junction of the upper and middle thirds of the stomach. (See diagrams.)

Although this chapter is mostly concerned with the diagnostic value of radiography, it should be mentioned that recently, both in the lay and medical press, much attention has been directed to a newer form of *radiotherapy* for carcinoma. For the past seven or eight years the medical electrician has been striving to manufacture an apparatus producing X-rays of very high penetration, resembling the *gamma* rays of radium. This seems now to be an accomplished fact. The newer method of radiation is superior to the older, as it gives a much greater homogeneity to the bundle of rays used. Wintz, of Erlangan, has elaborated a technique whereby the full dose necessary for the destruction of the diseased cells is given at one sitting. To him and his co-workers is due the credit of having introduced a *unit biological dose* with the percentage it is necessary to apply for the treatment of various pathological conditions—cancer, sarcoma, tuberculosis, etc. The results obtained by Wintz, at the Erlangan Clinic, have been most astonishing, and it is claimed that from 50 to 70 per cent. of uterine and mammary cancers have been cured. If further experience of the newer rays verifies the results obtained by Wintz it is quite certain that this form of radiotherapy will entirely replace operative treatment.

CHAPTER X.

GASTRIC LAVAGE.

This procedure is a most valuable therapeutic measure for many gastric derangements. It also often affords us much aid in diagnosis. There is a good deal of prejudice against its use. It is quite true that when it is employed for mechanical obstructions it can never be curative. For all such cases surgical interference is to be preferred to palliative lavage. But apart from such limitations it has a considerable field of usefulness.

A little practice makes the operation easy, and such discomfort to the patient as is inevitable at the outset of the procedure soon passes away, and most people readily acquire for themselves the art of passing the tube with a minimum of irritation. For the purpose of gastric lavage the simpler the apparatus, compatible with efficiency, the better it will fulfil the requirements. For most conditions the best time to carry out gastric lavage is at bedtime. This permits of a lengthened period of physiological rest to the stomach.

The ordinary stomach tube, used for the extraction of test meals, is quite suitable for this procedure. I have used for many years the tubes of Boas or Ewald with complete satisfaction. I prefer these patterns to the ordinary English tubes with closed ends and high lateral openings. A medium-sized tube is best suited for the purpose.

One end of a glass junction is fitted to the stomach tube, and the other end to an extension rubber tube which is attached to the funnel. The rubber tube leading to the funnel should be rather longer and have a slightly larger lumen than the stomach tube.

Although the glass junction is breakable, it has the advantage of supplying an observation window. Its lumen should never be less than that of the stomach tube. A slight shoulder near each end of the glass junction makes for security and tends to prevent

the stomach tube from parting company with the rest of the apparatus.

I use a funnel of aluminium of half a pint capacity. The glass funnel is too liable to breakage to be handled with the freedom desired in this procedure, but it has an advantage over the non-transparent funnels in showing the exact position of the irrigating fluid without lowering the funnel.

A Y-tube, with clamps, may be employed so as to enable one to use two fluids more readily. One limb passes to the receptacle for water and the other to that containing the antiseptic.

The patient should be seated on a chair with a high back, in the erect posture. The tube requires no lubricant other than that supplied by immersing it in hot water. The important thing is to have the rubber thoroughly warmed, as the introduction of a cold tube into the gullet is resented and excites such spasm as renders the operation difficult.

In introducing the tube the patient should be reassured that, however ' choky ' the sensation may be, there is not the slightest danger of suffocation. If the end of the tube be kept in close contact with the posterior wall of the pharynx, it is almost impossible for it to enter the larynx. It is just when it touches the pharynx that reflex spasm and retching occur and the patient becomes excited. If at this juncture he is sharply asked to swallow and then to take a long breath, and bend the head slightly forward, a gentle push will cause the tube to slip down the gullet without further trouble. If a sense of suffocation continues after the tube is in the stomach, it can be effectively relieved by asking the patient to take deep breaths. This manœuvre also facilitates the removal of the gastric contents.

When the tube has passed into the stomach the fluid should be poured into the receiving funnel, which is then elevated to the desired height. If it be raised too high too much pressure is developed and the mucous membrane might be injured by the inrushing fluid. Just before the funnel is quite empty it is quickly lowered into the receiving bucket and the longer column of fluid in the descending portion of the apparatus ensures syphonage.

For a great many purposes plain sterilised water may be used. As a rule it is best to introduce one pint of this and then to syphon it off. If an antiseptic solution is to be employed, it should then be introduced and in turn withdrawn. Lastly, another pint of

plain warm sterilised water is passed into the stomach to wash it clean.

Friedlieb's apparatus, with a bulb introduced at the position of the junction, permits of certain manipulations to be carried out. If the tube gets blocked, the obstructing material, by a brisk compression of the bulb, may be forced back into the stomach, and the tube cleared. If the column of fluid in the tube breaks, the bulb may be employed as an extractor to re-establish syphonage.

Lavage should be continued till the return is clear and free from solid debris. Over-distension should be avoided by introducing not more than 1 to 1½ pint of fluid at a time; but the condition for which this treatment is employed will determine the exact amount which it is safe to introduce into the stomach.

It is advisable to measure the amount of fluid returned. If much is retained we ought to seek an explanation for this. Serious discrepancies between the amount passed into the viscus and that returned might suggest hour-glass contraction.

The 'wash-out' should be examined macroscopically and microscopically. To obtain a suitable specimen the return from the stomach should be passed through a sieve and the solid material floated on a little water on an ashet. Any interesting particles should then be lifted by a mounted needle and placed on a slide for microscopic examination.

Various antiseptics may be employed to cleanse the stomach when there is retention of the contents and fermentation or decomposition going on. The choice will depend on the action desired. If lavage be employed to remove mucus, alkalies are used, while if it be used to stimulate this secretion, silver nitrate is indicated. The antiseptic and medicated solutions in common use may thus be tabulated:—

(1) *For removing mucus.*—This is an important part of the treatment of gastritis.

Solution of Borax (1 dram to the pint).

Solution of Sodium Bicarbonate (1 dram to the pint).

(2) *For stimulating mucus secretion.*—Often very helpful in the treatment of peptic ulcer.

Solution of Silver Nitrate (1 in 1000 or 2000).

To prevent any toxic action from retention of the silver solution, it is best to introduce only ten ounces at a time. When this has

been removed by syphonage, a second half-pint may be introduced. The stomach should be washed clean prior to the use of the silver solution.

(3) *For stimulating gastric secretion* (gastric juice).—Helpful in chronic gastritis and achylia.

Solution of Sodium Bicarbonate (1 dram to the pint).
Normal Saline Solution.

(4) *For diminishing over-secretion* (gastric juice).—Useful in hyperchlorhydria and gastrosuccorrhœa.

Magnesium Hydrate (Milk of Magnesium).
Bismuth Hydrate. Bismuth Carbonate.

These may be used alone, or combined, in sterilized water. Some Tincture of Belladonna may be incorporated in small doses (ten drops).

(5) *Antiseptic solutions for cleansing the stomach and inhibiting the growth of organisms and fungi.*—Useful in all conditions associated with stagnation and retention of gastric contents.

Sodium Hyposulphite (1 dram to the pint).
Borax (1 dram to the pint).
Sodium Salicylate (1 dram to the pint).
Permanganate of Potassium (gr. $\frac{1}{2}$ to 1, to the pint).
Milton (1 dram to the pint).
Boric Acid Solution (1 in 60).
Thymol Solution (saturated watery sol. mixed with equal parts of hot water).

(6) *To arrest hæmorrhage.*—Half pint of iced-water with 15-20 drops of Adrenalin solution.

(7) *To act as a sedative in pain and intractible vomiting.*—Lavage of the stomach is first carried out with warm normal saline solution. There is then left in the stomach, Nepente 15 drops, Hydrate of Bismuth 1 ounce, mixed in 3 ounces of warm gelatine solution. I have found this very effective. It may be repeated every three hours till relief is obtained.

Certain conditions contra-indicate the use of gastric lavage. The introduction of the tube into the unaccustomed gullet frequently induces retching and violent straining, and even though the flexible tube be employed the procedure is not always safe in certain diseases of the stomach, gullet, lungs or heart.

Thus the stomach tube should not be used in gastric ulcer after recent profuse hæmorrhage, hæmoptysis in phthisical patients,

cardiac disease with tendency to syncope or hæmorrhage, large aneurisms of the aorta and after-poisoning with the corrosive acids and alkalies.

On the other hand, lavage may be usefully and safely employed in the following conditions :—

(1) Peptic Ulcer with slight or no hæmorrhage. In this case an iced solution of Silver Nitrate (1 in 2000) slowly introduced into the stomach may give excellent therapeutic results.

(2) In all forms of dilatation of the stomach, especially if fermentation and decomposition are associated with the condition. Lavage is indicated not only in chronic but also in acute gastric dilatation.

(3) In chronic gastritis for the removal of mucus and in acute gastritis to allay persistent vomiting.

(4) In all cases of poisoning when the wall of the stomach has escaped serious injury. If this procedure is quickly carried out after poisoning, no other treatment yields results so satisfactory. Not only can the poison be extracted, but antidotes may be introduced which will destroy the action of any that may be left behind. Thus in opium and morphine poisoning, lavage with a weak solution of potassium permanganate is the treatment par excellence.

(5) In gastric tetany.

(6) Post-operative vomiting.

(7) During pregnancy.

Various modifications of the principle of gastric lavage have been introduced to meet certain therapeutic requirements. To carry these out, special instruments have been designed. Of these the more important are :—

(1) The Gastric Douche.—This form of gastric lavage aims at distributing the cleansing fluid over the entire mucosa without the risk of injury from over-distension or a too forcible jet of fluid.

The instrument required is nothing but an ordinary stomach tube, with many small apertures replacing the two or three large ones of the ordinary tube. The patterns of Einhorn and Rosenheim are very serviceable and efficient.

The gastric douche is best given on a fasting stomach.

Instead of the tube being passed well into the interior of the viscus, as for ordinary lavage, it should only penetrate through the cardiac orifice far enough to ensure that its apertures are free in the stomach. After the desired amount of fluid has been

introduced the tube is pushed well into the stomach and the fluid syphoned off. If a second douche is required, the tube is again raised and another supply of fluid administered.

Simple gravitation from a funnel I have found inadequate. To ensure the thorough douching of the entire mucosa some pressure is required. This may be provided for by means of a large rubber bulb syringe. Plain warm water or a medicated solution may be used according to the indications for treatment. Gastric douching may indeed be employed for all those purposes and conditions for which ordinary lavage is indicated.

(2) Gastric Spray (Einhorn).—Einhorn has invented this instrument, which aims at introducing fluid in a fine spray from a terminal nozzle. The common spray apparatus with a rubber pump and air bulb is employed. It has been recommended chiefly where only small quantities of medicated fluid are desired to be introduced into the stomach. Thus it may be used therapeutically, in peptic ulcer, to introduce silver nitrate solution to promote mucous secretion and cicatrization.

(3) Powder Insufflator (Einhorn).—To Einhorn we are also indebted for the intra-gastric powder-blower. By means of this instrument Bismuth and Magnesium Carbonates, Orthoform, and powdered dried supra-renal substance have been so administered in peptic ulcer. It is, however, very doubtful if this more complicated method offers any advantages over oral administration.

CHAPTER XI.

EXAMINATION OF THE SALIVA AND VOMITUS.

In many instances much valuable information can be gathered from an examination of the saliva and the vomitus.

Saliva ought to be secreted in sufficient amount, and with enough ptyalin in it, to make it active as a digestive fluid. For investigation as to its activity a small piece of chewing-gum aids its secretion, and enables a sufficient quantity to be collected in a porcelain saucer.

The reaction should first be taken with litmus paper. Rosalic acid paper, of a pale salmon tint, can be employed to ascertain, in one immersion, whether the saliva be alkaline or acid. If alkaline, the paper is turned bright red ; if acid, it becomes yellow.

In certain gastro-intestinal diseases the saliva becomes altered in its reactions.

The quantity of the saliva secreted may be diminished or increased. In most febrile conditions, diabetes (insipidus and mellitus), and atropine poisoning, there may be a marked reduction in the secretion. In chronic gastritis the secretion is often excessive.

There can be no doubt that important functional gastric derangements may arise from disorder in the salivary secretion, and more particularly from defects in its diastatic power. It may well be that amyl-dyspepsia, more or less complete, has its origin not in the stomach but in the mouth. Before it is assumed that gastric symptoms, due to failure of digestion of the carbohydrates, are referable to a hyperchlorhydria, the ptyalin activity should be ascertained. A few cubic centimetres of filtered saliva should be collected in two clean test tubes. An equal quantity of a tepid one per cent. starch solution should be added to each, well shaken with the saliva, and placed in an incubator, at the usual temperature of 37° C., for about ten minutes. Both tubes are then tested, one

with a drop of Lugol's solution, and the other with Fehling's solution, for sugar.

The iodine reaction will indicate the amount of starch changed, and roughly estimate the activity of the saliva in ptyalin. If the iodine stains a deep blue colour (unchanged starch) there is little or no ptyalin ; if a burgundy colour, digestion of starch is incomplete (erythrodextrin), and ptyalin is defective in activity or amount; while if there is only the slight yellow tinge, due to the drop of iodine itself, but no change in the colour, digestion is complete (achroödextrin).

By the Fehling test a quantitative estimate is possible.

In certain conditions not directly connected with diseases of the stomach the saliva may be usefully examined for certain abnormalities.

Fenwick has pointed out that the sulpho-cyanides, normally found in saliva, disappear when bile is prevented from entering the intestinal canal, and that in obstructive jaundice these reappear when bile re-enters the gut.

On a white porcelain slab place a drop of saliva. Bring it into contact with another drop of acid ferric chloride solution, when a pink colour will appear at the junction of the two fluids and ultimately permeate the entire commingled drops. The absence of this reaction indicates the absence of the sulpho-cyanides.

In diabetes the saliva often contains acetone, which may also be found in certain gastro-intestinal affections and various auto-intoxications.

Urea is also a commonly found abnormal ingredient of the saliva in chronic renal affections. Both it and acetone can readily be detected by the tests commonly used for their detection in urine.

In certain conditions the saliva may be required to be examined for micro-organisms.

The Vomitus.—This should be subjected to (*a*) macroscopical, (*b*) microscopical, (*c*) chemical and, if need be, (*d*) bacteriological examination.

(*a*) Macroscopic Examination.

(1) *Odour.*—Much will depend on the period of vomiting, so far as the smell is concerned. Food returned shortly after its ingestion ought to have only the odour peculiar to the ingredients which formed it. Later, when digestion may be advanced, a distinctly

'acid' odour is detectable. This acid odour of normal digestion is not disagreeable, but there is another 'sour' smell which is, and which indicates decomposition or fermentation with the formation of organic acids.

Or the vomit may have a 'biliary' odour or a fæcal smell when intestinal regurgitation is taking place.

Uncommonly the odour may be that of sulphuretted hydrogen. This is found occasionally, with retention, in simple pyloric stenosis.

(2) *Quantity.*—It is always important to note whether a vomit is only composed of the last meal taken, or whether it contains remnants of a previous meal or meals. Is the amount greater than that just taken? If so, one of two things may be assumed :—

(i) either that the stomach is emptying improperly; or

(ii) that there is hyper-secretion.

In certain neurotic affections, gastroptosis and dilatation, the vomitus is very large—due to retention.

(3) *The Appearance of the Vomited Material.*—Can undigested portions of meat be seen? If these occur in the vomit, after they have remained in the stomach for a time sufficient for their digestion, it is obvious that there is a fault in the gastric digestive power. It will probably be found, in such cases, that hydrochloric acid and pepsin are defective or absent. Starchy foods, such as white bread, are usually unrecognisable three hours after ingestion. If recognisable, as macerated portions of bread, there is also faulty gastric digestion. Delayed digestion of starches suggests hyperchlorhydria. If it can be established that particles of food taken in other meals, long before vomiting, are seen, then it is clear that we are dealing with 'delayed vomiting' from retention. This occurs in atrophic and cirrhotic chronic gastritis, in dilatation and gastroptosis, and most of all in organic obstructions of the pylorus from cancerous growth or simple cicatrization.

What is the *colour* of the vomitus? Normally it should coincide with the character of the food recently taken. Can any *red* blood be seen, or any altered by the digestive fluids and maceration to a brown colour—'coffee grounds'? The quantity of blood should be carefully noted. In ulcers inside the stomach bleeding is often large in amount. If this is the case it is quickly vomited, and has little time for change, and so comes up red. When only a small ooze has taken place, the stomach may tolerate its presence for a considerable time, and if vomited it is brown or blackish in colour.

The smaller bleedings (coffee grounds) are more typical of carcinoma than of peptic ulcer. Such small bleeding is often unrecognised by the patient, and if no vomiting occurs it may be wholly unsuspected, and can only be detected in the fæces as ' occult blood.'

Black vomiting (tarry) is seen as a terminal feature in some forms of peritonitis.

Bile in the vomitus, if present in any quantity, gives to it its characteristic greenish colour. The presence of bile indicates a regurgitation from the duodenum. When biliary vomiting is persistent, and is accompanied by the contents of the bowel, obstruction below the duodenum may be surmised. It should be borne in mind, however, that when retching is violent, bile may appear in the vomitus irrespective of the condition which causes the sickness. It is frequently seen in peritonitis, while the stomach contains no food at all.

Mucus.—Normally only a small amount should be present. In certain abnormal conditions the secretion is excessive. An abundant supply of ropy mucus is characteristic of one of the forms of catarrh—*e.g.*, chronic catarrhal gastritis. Sometimes an excess is found in achylia, although not commonly in the benign variety. It is almost always present in abundance in malignant achylia.

According to Boas, the vomiting of pure mucus, unmixed with food particles, is diagnostic of the absence of dilatation of the stomach.

Some of the mucus found in the vomitus may be derived from the mouth and gullet, and is swallowed with the saliva which is always more or less present.

Froth.—If much froth or air-bell be present, it usually indicates fermentation.

Pus is more commonly microscopic than macroscopic. It is found in some ulcerating cancers and phlegmonous gastritis, but it may be a contamination and not derived from the stomach itself. Many patients habitually swallow purulent secretions from the pharynx or from the pulmonary tract.

Decomposed or Fermented Vomitus is significant of some special abnormality. Hydrochloric acid plays a very important rôle as an antiseptic in the stomach. It is our first line of defence against organised poisons, which we may swallow with our food. Decomposition and fermentation denote either that there is great retention

of the food (Gastrectasis), or that there is little or no hydrochloric acid present. In these cases, in addition to the altered odour already referred to, such a vomitus will have a diagnostic appearance. It is frothy and usually abundant. When laid aside, in a glass cylinder, it will be noticed that it separates characteristically into three layers. At the bottom a precipitate, greyish in colour, of food debris ; an intermediate portion of fluid, more or less turbid ; and above, a layer of frothy mucus, in which are entangled the organisms causing the fermentation. In simple cicatricial stenosis, from ulceration, abundant hydrochloric acid may be present with an active growth of sarcinae ventriculi.

(4) *Parasites*, such as round worms, are occasionally seen in the vomited material.

(*b*) **Microscopic Examination of the Vomitus.**—Owing to the variety of materials of which a meal consists no very reliable data can be gathered from this investigation. Certain information of considerable value may, however, sometimes be obtained.

The presence of remnants of meat fibres in great excess, undigested starch granules, organisms, blood, etc., can be readily detected.

The microscopic examination of the expressed or extracted test meal is more informing, and the reader is referred to this subject in the paragraphs dealing with the examination of the test meal.

(*c*) **Quantitative Chemical Examination of the Vomitus** for hydrochloric acid and other ingredients is fallacious. Various contaminations, which vitiate the results, may find their way into the vomitus from the nose, throat, and mouth.

But the chemical examination of an expressed or extracted test meal is of great importance, and will be fully dealt with later on.

(*d*) **Bacteriological Examination** of the vomitus may also be misleading. Certain organisms, which might be regarded as pathogenic, may be derived from the upper passages during the acts of emesis or deglutition. To make a correct bacteriological examination the contents of the stomach must be syphoned off uncontaminated.

CHAPTER XII.

TEST MEALS.

A.—Technique.

Various forms of test meals may be given—the test 'breakfast,' 'dinner,' or 'supper' being employed, according to the results we desire to obtain.

Gastric secretion normally commences whenever food passes into the stomach, and digestion goes on till the chyme leaves the organ.

A test breakfast must be extracted one hour after its administration, while a test dinner must remain in the stomach for three to four hours.

The breakfast is largely starchy material, with only a limited proteid content which can utilise and combine very little hydrochloric acid. Thus in a short time the affinity for hydrochloric acid of the proteid material is satisfied, and the further secretion normally contains an abundant supply of free acid.

The dinner, rich in proteid, requires a much longer period before any hydrochloric acid remains free. The proteid can utilise or combine all that is secreted during the first two or three hours.

For most purposes the employment of a test supper and breakfast will suffice, but for certain investigations it may be necessary to give the test dinner.

As a routine the following practice yields excellent results :—

(1) **Test Supper.**—At bed-time some light meal may be taken—a little gruel, with a couple of teaspoonfuls of common currants or cranberries. These are given to test the motor power of the stomach and to ascertain if the pylorus is patent. The currants may be given, instead of with the supper, five or seven hours—or at any other fixed time—before the breakfast, if an exact estimation of the period required for the stomach to empty itself is important. The number of currants or cranberries or any remnants which may

be removed with the breakfast the following morning will give an indication of the gastric motility and of the condition of the pylorus.

(2) **The Test Breakfast.**—(*a*) *Ewald's.*—A slice of white bread about one quarter of an inch in thickness, with the crusts removed, should be toasted to a biscuit colour. If charred, or toasted too darkly, it gives a fictitious ' coffee ground ' appearance to the gastric content. With the toast, ten to twelve ounces of hot water or freshly infused weak tea are taken. The bread should be eaten dry, so as to ensure thorough mastication and fine division, and a mouthful of tea may be taken between each period of mastication. It is probable that tea is more stimulant of secretion than plain water, and may produce a higher percentage of hydrochloric acid, but it has the advantage of being more agreeable, and it more resembles an ordinary breakfast.

(*b*) *Boas' Breakfast.*—Two tablespoonfuls of oatmeal are boiled with two pints of water to one pint. A small quantity of salt is added. The Ewald test breakfast may add minute quantities of lactic acid to the extracted gastric contents, but this is avoided when the oatmeal is employed.

Occasionally it may be impossible, owing to the patient's condition, to give these meals in such quantities. We must then be content with a smaller meal.

Undoubtedly the best time to give a test breakfast is in the early morning. As far as this is possible, it is better not to tell a nervous patient that it is proposed to pass the stomach tube. I have observed in many cases with a normal digestion that the anticipation of the ' operation ' has sufficed temporarily to paralyse the gastric digestion and to disable the stomach functions. False results may thus be obtained. The ' stomach pump ' bulks largely in the imagination of the nervous invalid. By the exercise of a little tact the meals can be given and the extraction made without any of these drawbacks.

The Test Dinner (Leube-Riegel).—The dinner should consist of a plate of beef tea, a minced beef steak, mashed potatoes, and a roll of wheat flour. Such a meal should leave the normal stomach not later than five hours after its ingestion. To find some remnants of such a meal after the lapse of six hours is, however, no proof of abnormality ; but if considerable quantities of the dinner can be removed from the stomach after six, and more especially after eight, hours, we may be certain of the existence of one of the conditions

associated with loss of motility or of an interference with the outlet of the stomach.

If *achylia* is repeatedly found after the test breakfast, before we definitely make the diagnosis of suppression of gastric secretion, a further examination, by means of the test dinner, should always be undertaken to verify the condition. It may often happen, in certain conditions, that although the test breakfast failed to stimulate the gastric secretion, the nitrogenous test dinner may do so.

Stomach Tubes.—The best stomach tubes on the market are those with an open end and with lateral openings. They permit of better extraction and are more easily cleaned than those which have the end closed.

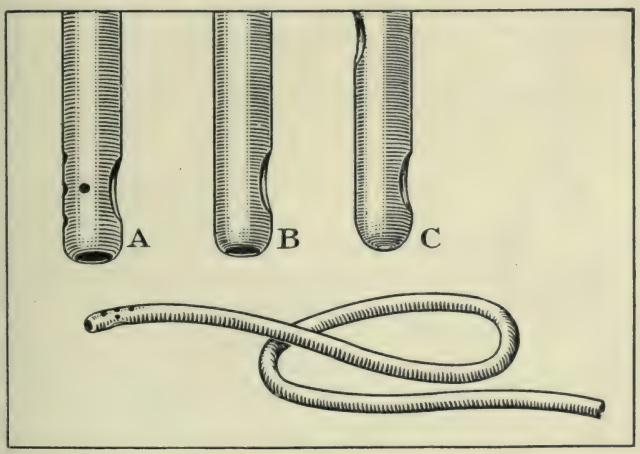


FIG. 16.
STOMACH TUBES.
A. Ewald's tube. B. Boas' tube. C. English pattern.

A good-sized tube which will permit of the easy passage of a swelled currant is essential for obtaining the best results. The patterns of Boas and Ewald are those which best fulfil the requirements. The ordinary English tubes with closed ends and two lateral openings are not good for several reasons. One of the openings is usually too far up the tube, and when it becomes uncovered syphonage or extraction will obviously cease even though all the contents may not be removed from the stomach. The closed end

also tends to retard the currants or other solid material from passing into the tube and thus gives an erroneous idea of the stomach contents. Nor is the English pattern so easily cleansed.

The meal is withdrawn from the stomach by one or other of the following methods :—

(1) *Simple Expression.*—This method consists in the utilisation of the patient's own efforts of expulsion, by contraction of the abdominal muscles and diaphragm, aided by the involuntary contraction of the stomach due to reflex irritation of the tube. The retching which commonly accompanies the introduction of the tube, in those unaccustomed to it, is often a most efficient aid to the expulsion of the test meal. If any difficulty be experienced in obtaining the meal, a sudden movement of the tube will often initiate the movements of vomiting and make the stomach contents pass up the tube. I never employ this method, because it often entails considerable straining, and in cases of erosion it may even be dangerous. It will be noticed that when straining has been severe, the last portion of the meal is apt to become blood-stained.

If this method be employed, there ought to be attached, by means of a glass junction, another piece of rubber tubing somewhat longer than the stomach tube itself. Once the chyme is driven the length of the descending tube, gentle syphonage results from the weight of the fluid in the longer limb, and the whole process is thus greatly facilitated.

At best, however, simple ' expression ' often results in failure. It is difficult, where active motility is present and the chyme passes quickly out of the stomach, to get any useful return by this method. In any case, we are never sure that we have obtained the entire contents. As it is often important to ascertain the amount present, any method which limits our knowledge of how far the stomach is normal, in its motor and secretory functions, must be condemned.

(2) *Extraction.*—There are many forms of ' pumps ' or extractors on the market. Some of them are complicated, expensive, and difficult to keep in order. The simpler the apparatus is, compatible with efficiency, the better it serves the purpose.

An essential in any extractor is that it be not too powerful. Small portions of mucosa may be aspirated off by using too strong suction. The danger of such a trauma need not be dilated on.

Sometimes the vacuum bottle of Potain may be employed, with intermediate rubber tube attached to it and the stomach tube. The metallic connexions are rather small, and apt to get blocked by solids.

Nothing answers better than a large rubber ball, fitted with easily detachable vulcanite nozzle of sufficient calibre to offer no obstruction to the gastric content, even though it contains solid particles of considerable size. Such an extractor as that of Kuttner, and employed by Ewald, answers all the requirements. The bevelled vulcanite nozzle, which fits into the screw top of the bulb, should be attached to the end of a piece of rubber tubing of the same calibre as the stomach tube. This tube is then attached to the stomach syphon by a good-sized glass junction, as described in the method of "simple expression."

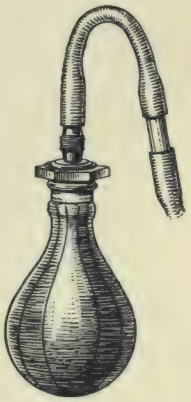


Fig. 17.
Kuttner's Extractor.

After the stomach tube has been passed into the stomach the bulb is then compressed and the vulcanite nozzle inserted. Gentle aspiration is thus effected. The advantage of the glass junction is obvious, for it permits the observer to see if the chyme is flowing freely. If not, gentle movement of the stomach tube—its partial withdrawal, its further insertion or rotation—should be practised. As often as the bulb expands it should be detached from the vulcanite nozzle, emptied, recompressed, and again attached until all the gastric content be extracted. If the tube becomes blocked the obstruction can easily be displaced by a sudden compression of the expanded bulb. A forcible current of air is thus driven through the tube. Care should be exercised not to exert more suction than is actually necessary. Too much may cause the invagination, into the end or lateral opening of the tube, of the mucosa, and prevent efficient extraction or remove a small portion of the mucous membrane.

The patient ought to be reassured that the procedure is not an operation, and that it will produce no pain, and can be carried out with perfect safety.

Glycerin may be used as a lubricator, but it will be found that

nothing more is necessary than to have the tube warm and wet by soaking it for a few minutes in hot water prior to its use.

The patient should be, as a rule, in a sitting posture. If there be any difficulty in extracting the test meal in this position, it is frequently immediately overcome by making the patient lie down on his right side. The tube should be passed slowly, till it reaches the pharynx, and then quickly be pushed into the gullet. It is just at this stage, when spasm and retching are apt to come on, with a sense of suffocation, that a little encouragement and the command "take a long breath" reassures the patient, and demonstrates to him that he is not choking. Deep breathing, in addition to removing the sense of choking, has the advantage of increasing intra-gastric tension during inspiration, and facilitates syphonage.

The tube should be passed about eighteen to twenty inches, less or more, according to the stature of the individual.

The employment of *fractional gastric analysis*, as introduced by Rehfuss, promises to be very useful in following and elucidating the digestive process in the stomach.

After the fasting stomach is emptied of any contents the patient is made to swallow a perforated metal bulb before the test meal is given. Into, and attached to, the bulb is a fine rubber tube, the end of which hangs out of the patient's mouth. The test meal is then given, and every fifteen minutes thereafter a small sample of the stomach juice, accumulated in the bulb, is aspirated by means of a suction syringe. The process is continued till digestion is completed. The amount of free HCl secreted can thus be accurately determined during the process of digestion, and graphically represented by a curve. This method shows that both in gastric and duodenal ulcer there is hyperchlorhydria and gastrosuccorrhœa.

B.—Examination of the Extracted Meal.

Macroscopic Appearances.—Very accurate information can be obtained by carefully studying the physical characters of the returned meal. (See also Vomitus, page 105.) The following points will be noted :—

(1) *Its general appearance.*—After an Ewald's breakfast it should be homogeneous, smooth, and of a varying consistency, according to the amount of fluid with which it is mixed. Normally it resembles a purée of powdered biscuit. If the toast has been

properly chewed and well digested, no recognisable particles should be present.

When filtration is proceeded with, some information can be gained by observing the speed at which it passes through the filter paper and the appearance of the filtrate. When the returned gastric content contains much mucus the rate of filtration is very slow. When little or no digestive action has taken place, as in simple achylia, the filtrate is as clear as water, but when digestion has been complete the filtrate is usually turbid.

(2) *Quantity.*—The extracted contents should be measured. In many morbid conditions of motility and secretion the quantity varies greatly. Normally, in one hour after the test breakfast, the contents vary from 15 to 80 c.c.'s. The lower and the higher figures may, in certain circumstances, be regarded as abnormal. An average amount from a healthy stomach should be about from 30 to 40 c.c.'s. Many conditions co-operate to increase or diminish the return. If the contents of the stomach be abnormally large, it is well to set the entire quantity aside, in a glass cylinder, and observe how the meal behaves. In most cases of retention, with fermentation, the gastric contents separate into three layers. At the bottom an abundant layer of greyish finely granular material precipitates, then an intermediate layer of somewhat turbid fluid, and on the top a greater or lesser layer of frothy mucus and debris.

Increase may be due to hyper-secretion (magensaftfluss) or retention (atonic or stenotic). If hyper-secretion be suspected, it is well to empty the stomach completely at bedtime, and the following morning to syphon again before any food of any kind has been taken.

True hyper-secretion should only be diagnosed when considerable quantities of pure gastric juice can be extracted from a stomach unstimulated by food.

If gastric retention be present, enormous quantities may be returned if the stomach has not been emptied before the test meal has been given. It is obvious that an excessive return from the stomach, which normally absorbs very little water, can only be accounted for by hypersecretion, or by retention. In the latter case one often gets back almost the entire amount of fluid which formed part of the test breakfast. It often happens, too, in dilatation and retention from pyloric stenosis, that the amount

returned far exceeds the total volume of the test meal. This is due to an addition from previous meals.

Decrease.—This may occur from hyper-motility and hyper-acidity with a normal pylorus, as these conditions favour rapid emptying of the stomach. It is also found that an abnormally small amount of the test breakfast may be returned in some cases of achylia, gastrica nervosa, and other benign forms of this disorder from abnormal relaxation of the pylorus.

(3) *Colour.*—Note if bile be present, or streaking or tinging with red blood can be seen. Any colour other than normal ought to be noted, and its cause investigated later. A dark colour like tea or coffee may suggest altered blood.

(4) *Smell.*—The extracted breakfast ought to have no unpleasant smell. A sweetish odour, of pepsin-like character, should alone be detectable. A sour, acrid, rancid, or decomposed smell is always abnormal. The presence of sulphuretted hydrogen, and the barmy or beer-like odour of fermentation, may have special significance.

(5) *Froth and Mucus.*—If much froth be present, it usually indicates fermentation. Mucus is present normally in small amount only. If excessive it indicates catarrh. It is rarely found in gastric ulceration.

(6) *Reaction.*—Litmus paper may first be employed to test the reaction.

Having obtained a sufficient amount of gastric juice, the following routine examinations should be made :—

Qualitative Tests for Free Hydrochloric Acid.—(1) *Congo Red.*—This may be employed as a liquid reagent or, much more conveniently, in the form of a paper.

It should be noted that litmus paper is not diagnostic of the kind of acid present. Blue litmus paper will usually be turned red, even though no free hydrochloric acid be present, owing to the readiness with which organic acid may form in the absence of hydrochloric acid. The intensity of the reaction to litmus should, however, be noted.

If a few drops of a weak watery solution of congo red be added to a small quantity of the filtrate, in a porcelain saucer, the presence of free hydrochloric acid may be demonstrated at once by the change from red to a blue colour. The same reaction will be observed by

immersing one end of a small strip of the red paper* in the filtrate. The purity and the depth of the blue colour must be carefully noted. When experience of the use of congo red paper has been gained it can almost be trusted to approximately estimate the amount of free hydrochloric acid present. The red colour is replaced by a light to a very deep indigo-blue colour, according to the amount of acid present. When free hydrochloric acid is defective, organic acids are apt to be developed. A mixture of free hydrochloric and organic acid changes the red to a brown or dirty blue. In the entire absence of hydrochloric acid and organic acids no change takes place in the colour of the congo red. It should be observed that congo red will turn deeply blue in presence of very large quantities of lactic acid. As the amount of this acid sufficient to produce this reaction is very rarely found in any gastric content, congo red becomes, after some experience, a diagnostic test for free hydrochloric acid.

(2) *Dimethylamidoazobenzol.*—Similarly to congo red, dimethylamidoazobenzol may be employed in a solution or in the form of a test paper.* The latter is much more convenient, and, with some experience, the amount of free hydrochloric acid present may also be roughly estimated by the depth of the red colour produced on immersing it in the gastric filtrate.

Dimethylamidoazobenzol in weak solution is of a canary-yellow—in stronger solution of a pinkish-yellow—colour. Either the solution or the paper is turned to a coral-red of varying depth, according to the amount of free hydrochloric acid present. If none be present there is no change in the colour. A combination of organic and free hydrochloric acids will cause the coral-red to assume a rather dirty colour with a brownish tinge.

(3) *Resorcin-Sugar Test* (*Boas' Reagent*).—The formula for this reagent is :—

Resorcin	5 grams.
Sugar....	3 ,,
Alcohol	100 c.c.

Equal parts (a few drops) of the reagent and gastric filtrate to be tested are put into a small porcelain saucer and slowly evaporated

* Congo red and dimethylamidoazobenzol papers may be economically and conveniently made by immersing white filter paper in a weak watery solution of congo red (gr. iij—℥i) and an alcoholic solution of dimethylamidoazobenzol (gr. x—℥i) respectively, hanging the sheets up to dry and cutting the papers into convenient sized strips.

to dryness over a small spirit flame—great care being taken to prevent charring. In presence of free hydrochloric acid a bright vermilion-red colour will be produced, which fades on standing.

The test is a very reliable one for free hydrochloric acid.

(4) *Phloroglucin-Vanillin (Günsburg's Test).*—This reagent, which contains rather expensive ingredients, and which does not keep well,* consists of :—

Vanillin	1 gram.
Phloroglucin....	2 grams.
Absolute Alcohol	30 „

An equal number of drops of the filtrate and reagent is dealt with in the same way as in the preceding resorcin test. The reaction is positive for free hydrochloric acid if, on evaporation, a cherry-red colour is produced. A yellowish-brown tint indicates an entire absence of free hydrochloric acid.

(5) *Tropeolin OO Test.*—A few drops of a saturated alcoholic solution of tropeolin OO are put into a porcelain saucer. This reagent has a yellowish-brown colour which, in contact with hydrochloric acid, turns red.

If carried out as recommended by Boas, tropeolin OO is a very delicate test for free HCl. A few drops are spread over a porcelain saucer and an equal quantity of the gastric filtrate is allowed to flow over the same area. Gentle heat is applied by means of a small spirit flame. In presence of free HCl lilac streaks develop at the edges, which on further heating turn blue.

Test papers of tropeolin OO can be readily made by saturating white filter paper in the alcoholic solution, drying and cutting into suitable strips.

Quantitative Tests for Hydrochloric Acid.—Having demonstrated the presence of free hydrochloric acid in the gastric content, the next step in the examination must be the estimation of the amount present—*i.e.*, a quantitative test.

Hydrochloric acid is present in the test meal, in the normal stomach, free and combined. A test breakfast composed, as it largely is, of starch, has no great affinity for hydrochloric acid. The smaller proteid content rapidly combines with the hydrochloric acid as it is secreted until its affinity is satisfied. All that

* If the solutions of phloroglucin and vanillin be kept separately, and mixed in equal quantities at the time of use, the reagent is prevented from deteriorating.

is secreted over and above the amount combined by proteid remains as free hydrochloric acid. Thus it is that only one hour is required to elapse before a test breakfast is removed for examination. With a test dinner rich in proteid, " combination " of free hydrochloric acid goes on for a lengthened period, and four to four and a half hours are required to elapse before extraction of the meal for examination. If removed too soon, obviously an abnormally low reading of free hydrochloric acid will be found.

The combined hydrochloric acid may be neglected in cases where free hydrochloric acid is found in normal amount. But in certain conditions it is essential to estimate its amount, both for diagnostic and therapeutic purposes. Thus, in cases of achylia, before concluding that no hydrochloric acid is being secreted by the stomach, combined acid ought to be sought for.

The total acidity is much more important, and should always be ascertained. In estimating the total acidity it must ever be kept in mind that we are dealing with (1) free hydrochloric acid, (2) combined hydrochloric acid, (3) varying amounts of organic acid, and (4) acid salts (phosphates).

It is quite true, however, that for clinical purposes, when a normal or abundant free hydrochloric acid content is found, we may disregard the organic acids. The very presence of a normal amount of hydrochloric prevents the formation of any appreciable quantity of organic acid.

For these estimations the *Töpfer's method*, or some modification of it, is the best.

A number of reagents are required :—

(1) 1 per cent. solution, in alcohol, of Phenolphthalein.
(2) 5 per cent. solution, in alcohol, of Dimethylamidoazobenzol.
(3) 1 per cent. solution, in water, of Sodium Alizarin Sulphonate.
(4) A decinormal solution of Sodium Hydrate.

In this and other methods for estimating the acidity of the gastric content colour reagents, which react definitely to hydrochloric acid, are employed as " indicators."

Method.—Ten cubic centimetres of gastric filtrate (if sufficient has not been extracted five c.c. may be used) are placed in three glass beakers numbered 1, 2, and 3.

No. 1.—To this beaker add two drops of dimethylamidoazobenzol, the " indicator " for free *hydrochloric acid*, and mix it thoroughly with the gastric filtrate. The presence of hydrochloric acid is

indicated by the immediate change of the yellowish solution into a bright red-currant-juice colour. The depth of the red colour is somewhat dependent on the amount of hydrochloric acid present.

No. 2.—To this add two drops of phenolphthaleine solution. This colourless solution is the indicator for *total acidity.*

No. 3.—Two drops of a solution of sodium alizarin-sulphonate are added to this beaker. This is the reagent for combined hydrochloric acid. It is dark brown in colour, and slightly darkens the gastric filtrate to which it is added.

The Estimation.—Having thus prepared the three portions of gastric filtrate a graduated burette is filled with one-tenth normal sodium hydrate solution (4 grams sodium hydrate to 1000 c.c. distilled water). Note the level of the sodium hydrate solution in the burette before and after the completion of each reaction, and thus the number of c.c.'s required to effect complete neutralisation.

(1) *Free Hydrochloric Acid.*—To the gastric filtrate, coloured red by the indicator (dimethylamidoazobenzol), the sodium hydrate is added drop by drop with constant trituration until the *end reaction* is reached. Complete neutralisation of the free hydrochloric acid is indicated by the formation of a lemon or canary-yellow colour of varying degrees of purity. If no appreciable quantity of organic acid be present, a pure lemon-yellow tint will result, but when in addition organic acid is present a rather dirty yellow or brownish yellow tint results.

One cubic centimetre of decinormal sodium hydrate solution is equivalent to, and therefore neutralises, 0.00365 grams of hydrochloric acid. Thus, if we have used, say, 3.6 c.c. of decinormal solution, the amount of hydrochloric acid in the 10 c.c. of gastric filtrate must be $0.00365 \times 3.6 = 0.01314$ gram. The percentage would thus be $0.01314 \times 10 = 0.1314$.

(2) *Total Acidity.*—When the phenolphthaleine solution is added to the gastric filtrate, if acids be present, there is no change in colour.

The sodium hydrate solution is added in the same manner as above until a permanently red colour, which does not vivify on the further addition of sodium hydrate, is obtained.

The total acidity may be conveniently expressed by the number of cubic centimetres of sodium solution required to produce the end reaction.

If 8 c.c. of the decinormal solution be required to neutralise 10 c.c. of gastric filtrate, ten times that amount will be required to neutralise 100 c.c. Thus, $8 \times 10 = 80$ degrees of acidity. Or this may be expressed in percentage of hydrochloric acid by multiplying the number of cubic centimetres by 0.00365—*i.e.*, $8 \times 0.00365 = 0.2920$.

When free hydrochloric acid is present in normal amount there is almost always a uniform relationship in the reading to that of the total acidity.

The following table gives *approximately* my results :—

FREE HYDROCHLORIC ACID.	TOTAL ACIDITY.
0.14 per cent.	60
0.18 ,, ...	70-75
0.214 ,, ...	80-90

Any striking discrepancy between the free hydrochloric acid and the total acidity should be noted. In sub-acidities the antiseptic action of the gastric juice is less potent, and it is in these cases that organic acids are apt to form in lesser or greater proportions. Thus we sometimes find a hypochlorhydria and a high total acidity may be associated. This suggests at once the presence of an abnormal amount of organic acid.

(3) *Combined Acid.*—It will be observed that the sodium hydrate solution is added till neutralisation of the free hydrochloric acid, organic acids, and the acid salts (phosphates), is obtained.

Proceed in the same way as for the estimation of free hydrochloric acid. The amount of decinormal solution required to produce the end reaction is noted. For beginners who are not familiar with the exact colour of the end reaction a standard colour may be produced by the addition of three drops of alizarin solution to five c.c. of a one per cent. solution of sodium carbonate. This end reaction—a violet colour—indicates the neutralisation of free HCl, organic acids, and acid phosphates—the remaining acidity of the gastric filtrate being due to combined hydrochloric acid.

The amount is estimated thus :—

If 5 c.c. of decinormal solution of sodium hydrate be required to produce the violet end reaction in 10 c.c. of gastric filtrate, it would take 50 c.c. for 100 c.c. of gastric filtrate. Multiply the number of c.c. of decinormal solution by the amount of hydrochloric acid required to neutralise one c.c. of sodium solution—thus, $50 \times 0.00365 = 0.1825$ per cent., which is the amount of

acidity due to free hydrochloric acid and acid salts. Suppose the total acidity to be 0.2555 per cent. Subtract from this the percentage of free acid and acid phosphates—thus, 0.2555 − 0.1825 = 0.0730 per cent. of combined hydrochloric acid.

From these data it is obvious we can also calculate the amount of organic acid present. If we add the free hydrochloric acid to the combined hydrochloric acid, and deduct this figure from the total acidity, we shall be left with the amount of organic acid present.

Sjöqvist Test for Combined HCl in cases of Achylia.—Place 10 c.c. of filtered gastric juice in a small nickel crucible with a large pinch of Barium carbonate, evaporate to dryness and incinerate over a Bunsen flame. In the presence of HCl some barium chloride is formed—the only soluble salt of barium. Triturate the residue well with distilled water. Filter repeatedly till a perfectly clear solution is obtained. Transfer this to a test tube and add H_2SO_4) (1 in 3) or a solution of Na_2CO_3. A precipitate shows that there must have been some HCl combined with the albumin in the stomach filtrate. The reaction may thus be represented :—

$$\text{Alb.} < \begin{matrix} \text{Cl} \\ \text{Cl} \end{matrix} + BaCO_3 = BaCl_2 + CO_2 + O$$

The CO_2 escapes and the O is burned with the albumen.

This test is only useful, in cases where free HCl is absent, to determine if there be a *total suppression* or only a *deficiency* in the secretion of the acid.

Other and more simple methods of making these estimations may be carried out as follows :—

If Weiss' tube be employed, accurate enough data for clinical purposes can be obtained with ease and without any calculation. The tube, which has a double scale, is employed to estimate the free and combined hydrochloric acid and the total acidity.

(1) *For quantitative estimation of free hydrochloric acid* (Weiss' Tube).—Place 10 c.c. of gastric filtrate—that is, up to the mark M in the tube (or to ½ M, half meal, if there be not sufficient filtrate and add distilled water to make up to mark M). Add two to three drops of the double indicator of Linossier.* Then add in small

* Linossier's double indicator has this formula :—

Dimethylamidoazobenzol ..	0.25 grm.
Phenolphthalein	120 grm.
Alcohol (90%)	100 grm.

streams, and later on drop by drop, the decinormal sodium hydrate solution, inverting the tube between each addition, till the red colour, due to the action of the hydrochloric acid on the dimethyl-amidoazobenzol solution, is permanently changed to a yellow colour. If no further vivification of the yellow tint takes placeby the addition of another drop or two we have reached the end reaction—that is to say, the complete neutralisation of free hydrochloric acid. The amount of free acid is then read off, in percentage, on the scale at the right hand side of the tube. The reading is doubled if only half a meal has been used.

(2) *Total Acidity.*—By further addition of the decinormal solution the yellow colour will gradually pass from orange to red. This is due to the neutralisation of *all* acids and the slight alkalinising of the gastric filtrate, which then changes the colourless phenolphthaleine into a red-currant-juice tint. This end-reaction colour is identical to that produced by free hydrochloric acid and dimethyl solution. When this colour is attained, and the further addition of a drop or two of decinormal solution produces no vivification of the red colour, we again have reached the end-reaction. The height at which the fluid in the tube now stands is read off on the scale on the *left*-hand side. This indicates the total acidity.

(3) *Combined Acid* (Robins' Test).—To estimate the combined acid with Weiss' tube, Robins' method is the best. To the usual amount of gastric filtrate in the tube add two drops of a one per cent. alcoholic solution of hæmatoxylin. This indicator strikes yellow, and remains so as long as any free hydrochloric acid or organic acid is present.

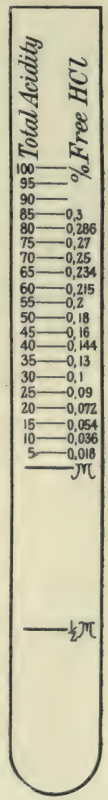


FIG. 18. WEISS TUBE.

Drop the decinormal solution into the tube, as carried out for the estimation of free hydrochloric acid, till the filtrate assumes a permanent blue colour on inversion of the tube.

As long as free hydrochloric and any organic acids are present the indicator remains yellow. The combined acid present does not affect its colour till the free hydrochloric and organic acids are completely neutralised. The end reaction is a blue colour,

remaining permanent on inversion of the tube. Note on the scale the figure indicating the degree of acidity, and subtract it from that of the total acidity to ascertain the combined acid.

In this calculation the acid phosphates have been neglected. If allowance is made for these, the amount of combined acid will be slightly lower.

Examination for Organic Acids.—The presence of these will have been detected, or suspected, by the foregoing investigations. The more important are lactic, butyric, and acetic acids.

The clinician need not make any attempt to estimate quantitatively, by chemical analysis, the presence of these substances. The character of the reactions will suffice to indicate whether there are only traces or considerable quantities present in the extracted test meals. Minute traces of these may be neglected as having no clinical significance.

(1) **Lactic Acid.**—Two tests may, with certain precautions, be employed for the detection of lactic acid :—

(*a*) Perchloride of Iron solution.
(*b*) Uffleman's reagent.

Both of these tests should be carried out with Strauss' separator. If the ferric chloride solution be added directly to the gastric filtrate, or the gastric filtrate to Uffleman's reagent, a number of fallacies may occur. Peptone, alcohol, and sugar, as well as some organic acids, give a positive reaction. As these substances are almost always present in some proportion in gastric contents, it will be seen that they must be first eliminated before we can assert, with accuracy, the presence of lactic acid. These ingredients can be excluded by making an ethereal extract of the filtered stomach contents in Strauss' separator. The test is carried out as follows :—

Strauss' Method.—Place in the separator five c.c. of gastric filtrate (up to the first mark), and on the top of it twenty c.c. of ether (up to the second mark). Shake vigorously. Allow it to settle till all the ether floats to the top. Open the stop-cock and run off the five c.c. of gastric filtrate. To the twenty c.c. of ether left add five c.c. of distilled water, shake again vigorously, and allow to stand till the water settles at the bottom of the separator. Open the stop-cock and run off the distilled water into a clean test tube. The ether extracts the lactic acid from the gastric filtrate, and leaves behind the substances which are apt to give fallacious

results. The water in its turn takes up the lactic acid. Add to it two drops of a watery solution of ferric chloride (1.9).

In the presence of lactic acid a canary-yellow-green colour of varying intensity, proportionate to the amount of acid present, will be struck. Strauss has a scale of tints indicating approximately the percentage of lactic acid present. With these tints, the colour struck in the test tube is compared to estimate the amount of acid present.

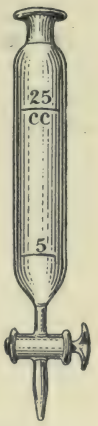


Fig. 19. Strauss Separating Funnel.

Uffleman's Test.—In carrying out the test the reagent should be made freshly. Into a clean test tube run ten c.c. of a two per cent. watery solution of acidum carbolicum liquefactum and one drop of liquor ferri perchloridi. This mixture turns to a clear amethyst-blue colour. It should not be too dark, but of a light clear colour.

Another method of producing the reagent is as follows :—

Mix ten c.c. of a four per cent. solution of acidum carbolicum with twenty c.c. of distilled water, and to this add one drop of liquor ferri perchloridi.

The reaction consists in the formation of iron lactate (yellowish-green in colour). The amethyst tint, produced by the addition of carbolic acid, is not essential to the test, but is used only to obtain a contrast colour.

As ordinarily carried out, by running a small stream of gastric filtrate into the reagent, the reaction is very unreliable, and unless lactic acid be present in large quantities, no dependence can be placed on this method. But if the extraction with ether be employed as described above, and the watery extract be added to the Uffleman's reagent, and a yellow colour results, the test is positive for lactic acid. The depth of the yellow colour produced, and the amount of filtrate required, will give a rough quantitative estimate of the lactic acid present.

Significance of Lactic Acid.—In normal digestion lactic acid is never found in appreciable quantities unless it be introduced with the food. In that case it would have no pathological significance. To be of diagnostic value it must be found in the extracted contents after a meal which contains none. Ewald's breakfast of bread or toast, with tea, is not quite lactic acid free. A trace, which has

no significance, may therefore be found in the contents extracted after this breakfast.

Boas recommends that about half a pint of well boiled gruel, served without milk, cream, or sugar, should be used. This meal may be made by boiling one ounce of oatmeal in one pint of water till the latter is reduced to half a pint. The stomach should be washed out the previous night to eliminate all source of fallacy. This meal is lactic acid free, and if any appreciable quantity can be detected in its filtrate the reaction may be regarded as pathologic.

In achylic conditions, where no appreciable quantity of lactic acid is apparently present, the gastric filtrate should be acidified, drop by drop, with dilute hydrochloric acid till it turns congo red to the normal blue colour. This preliminary step is necessary before we can assume that lactic acid is wholly absent, because in achylic and achlorhydric conditions, lactic acid, if present, will have ' combined ' with proteid. On addition of hydrochloric acid it is displaced, and can readily be detected by the appropriate tests.

Lactic acid is found in gastric contents in various conditions.

It is most commonly present in all cases of gastric stasis, with a low acidity (hypochlorhydria). In all forms of extreme motor insufficiency and pyloric stenosis lactic acid will usually be found. It is characteristically present in cases of gastric carcinoma, and often in large amounts. It results from fermentation of the carbohydrates due to the action of the lactic acid bacillus (Boas-Oppler). When lactic acid is present, these organisms will be found microscopically.

(2) **Butyric Acid** is a volatile fatty acid, very readily detected by its pungent and characteristic odour of rancid butter.

Mix in a test tube about five or six drops of gastric filtrate, with an equal quantity of alcohol, and add two c.c. of concentrated sulphuric acid. With the heat generated by the action of the sulphuric acid an agreeable odour of pineapple (Ethyl Butyrate) will be developed.

(3) **Acetic Acid** is also readily recognised by its smell of vinegar, if present in any pathological quantities. It can be chemically tested. To test tube containing ten c.c. of gastric filtrate add fifty c.c. of ether. Shake well together. Decant the ether and evaporate.

Dissolve the residue in distilled water. Add a few drops of phenolphthaleine indicator and neutralise with decinormal sodium hydrate solution. To this solution add a few minims of a dilute solution of ferric perchloride. If acetic acid be present a dark red colour will result.

Blood in Vomitus or Gastric Content.—Blood may occur in all quantities in the gastric contents. It may be obvious (macroscopic) or may only be detected by means of chemical or microscopic examination (occult blood).

Blood may be found in the vomitus or test meal in many conditions. Most commonly it is present in simple or malignant ulcerative processes.

It is also frequently due to venous congestion, when it may occur in cardiac disease and cirrhosis of the liver.

A number of cases of severe hæmatemesis and malæna find their way into my hospital wards, diagnosed as gastric ulcer, which are really due to cirrhosis of the liver and œsophageal bleeding. This cause of gastric hæmorrhage should not be overlooked.

In acute alcoholic and other toxic gastritis the vomitus is often more or less tinged or ticked with blood. Thus in the terminal phases of chronic Bright's disease, with uræmia, the vomitus frequently contains blood from erosions of the gastric mucosa. In addition to these more common causes, certain other conditions may be associated, without gross lesion, with bleeding from the stomach and other mucous tracts. Purpura, hæmophilia, pernicious anæmia, and the leukæmias, vicarious menstruation, and the obscure condition known as *gastrotaxis*, all may be associated with hæmorrhage from the gastro-intestinal mucosa.

The physician should not overlook the possibility of malingering. In hysteria a categoric history of hæmatemesises may be given, but should never be accepted without investigation.

A history of vomiting of blood during the night alone should always arouse suspicion as to its genuineness. Neurasthenics and malingerers adopt most ingenious plans to deceive their entourage, and not infrequently even impose on expert observers.

Blood from the gums, the juice of certain fruits as black cherries, black and red currants, and certain dyes, have all been employed to cause deception. Real blood may be collected from the vagina during menstruation, or expressed from the gums and mixed with saliva and some food and passed off as a hæmatemesis.

It is also well to remember that the strain of vomiting may produce slight laceration of some vessel, and occasionally bleeding. During syphonage, too, if great straining results, or if the extraction of the gastric contents requires much manipulation, a small quantity of blood, which has no clinical significance, may be found in the vomitus or test meal.

In all cases of alleged hæmatemesis the stools should be examined for macroscopic and occult blood.

Differential Diagnosis of Hæmorrhage.—Having determined that blood is really present in the vomited material our next effort must be to locate its source, and determine its cause.

The nose, mouth, and throat should first be examined, and any erosion or bleeding spot eliminated as a source of the hæmorrhage. As a rule the differential diagnosis of hæmatemesis from hæmoptysis is easy, although in certain cases it presents great difficulty. In slight pulmonary hæmorrhage all the blood may be swallowed and then vomited, mixed with the gastric contents. In such cases the diagnosis of ulcer may erroneously be made, especially as there may be a history of digestive derangement—the dyspepsia of phthisis. Such symptoms as cough, expectoration, hæmoptysis, emaciation and fever must be sought for and a careful pulmonary examination made. The patient should be interrogated as to whether the hæmorrhage followed a sudden faintness and pallor. In a profuse gastric hæmorrhage there is usually a history of sudden faintness and pallor prior to the actual hæmatemesis. In hæmoptysis the blood is usually bright red and frothy.

The macroscopic examination of the gastric content for blood.—When gastric hæmorrhage is large the blood is certain to be thrown up quickly and little altered; but when it is small in amount it is tolerated by the stomach for some time, and then undergoes change in colour and appearance. When vomited, the blood-contaminated gastric content resembles a brownish granular material not unlike hare soup. The colour may vary from light to dark brown (*coffee grounds*), or it may even be black. These changes are due to the action of the digestive fluid and stomach gases on the blood.

The larger hæmorrhages are usually found in association with gastric and duodenal ulcers, and the smaller with cancer.

In peptic ulcer hæmorrhage is apt to be intermittent, while small persistent hæmorrhage is more characteristic of malignant disease.

Far too commonly peptic ulcer is overlooked because the vomitus or stool apparently contains no blood. It is quite true that the entire absence of blood, as found by repeated chemical examinations, goes far to exclude ulceration, but it is frequently taken for granted that the vomitus of the fæces contain no blood because none is seen.

I have found that in at least seventy per cent. of all cases of peptic ulcer and advanced carcinoma occult blood is present either in the gastric content or fæces, or in both. Hence the importance in chronic gastric conditions of a search for occult blood.

It is an essential condition that if occult blood be found in the gastric content or fæces, as a sign of disease, that it be derived from the gastro-intestinal tract. The tests employed are so delicate that hæmoglobin from meat, or any medicine containing iron, must be excluded. In the case of the fæces the proper procedure is to give a hæmaglobin-free diet for three days and to test a portion of the motion passed on the fourth day. During this time no iron must be taken, as it gives a positive reaction for blood.

The following tests and methods are alike suitable for the detection of occult blood in the gastric contents and fæces. Among those many are simple and reliable, and the practitioner need only carry out the Guaiac test with the certainty that it will give accurate results if he observes the proper technique :—

(1) **Guaiac Test.**—*Reagent.*—(1) Place in test tube a very small pinch of powdered Guaiac resin.

(2) Dissolve it in five c.c. of 69 per cent. alcohol.

(3) Add an equal quantity of reliable and active peroxide of hydrogen, and mix thoroughly together.

(4) Set this reagent aside for a few minutes, and observe if any change in colour occurs. It should remain clear, and of a pale straw colour. The formation of any blue tint indicates some impurity or deterioration of the reagent—usually of the H_2O_2.

Technique.—(1) Place in a small mortar some of the fluid and solids of the gastric content, or a small portion of fæces.

(2) Add an equal quantity of glacial acetic acid.

(3) Triturate well with pestle for a few minutes.

(4) Transfer to a perfectly clean test tube.

(5) Add an equal quantity of methylated ether, and invert the tube briskly several times.

(6) Set aside for the ether to separate to the top of the tube.

(7) Float the ethereal extract on the top of an equal quantity of the reagent, taking care to let the extract flow gently down the inclined tube so as to get a delicate contact reaction.

(8) A blue ring at the junction is positive.

In this test the glacial acetic acid converts the hæmaglobin present into acetic hæmatin or hæmatin acetate, which is extracted by the ether.

It should be noted that any impurity in the peroxide of hydrogen will vitiate the result and give a positive reaction when no occult blood is present. It will be readily understood that a grave error may result if an erroneous diagnosis be made. I find the peroxide of hydrogen of Merck a good ozonic solution. It is highly concentrated, keeps well, and can be freely diluted with distilled water when it is required for testing.

Whichever peroxide solution is employed it is well to make a control test to determine its integrity. This is done by mixing an equal quantity of the peroxide solution to be used with the Guaiac solution and laying it aside for a short time. If no spontaneous blue colour appears in the solution, the reagent may be considered reliable. From time to time the validity of the test reagents should be verified by testing fæces or vomitus known to contain blood, or to which blood has been added for the purpose.

In testing the fæces it is essential to exclude any artificial contamination from piles, fissures, or from menstruation. It is always best to get a *solid* part of the prepared motion. This should be placed on a porcelain slab and a central portion cut out by means of a clean blade, to ensure the removal of any blood from the surface of the fæces. Such a portion of fæces giving a positive reaction indicates that the blood is an intrinsic part of the stool.

(II) **Aloin Test.**—This test is also very reliable. Its technique is identical to that of the Guaiac test. Substitute for a 'very small' pinch of Guaiac a large pinch of pure Aloin, and the procedure is identical. A positive reaction is a red colour or ring at the junction of the reagent and ethereal extract. A few minutes should be allowed to elapse before a negative reaction is admitted. The reagent may be made more delicate by diluting it with an equal quantity of spirit.

(III) **Teichmann's Hæmatin Test.**—This test, as modified by Professor C. Strzyzowski, is an easy and reliable one for the revelation of occult blood in the gastric contents.

The *reagent*, which must be prepared freshly for each reaction, consists of :—

Glacial Acetic Acid.
Alcohol.
Distilled Water, āā 1 c.c.
Hydriodic Acid, m. 3.

A few drops of the gastric filtrate, or any suspicious brownish grains, are placed on a microscopic slide and dried over the spirit flame. A cover-slip is then placed over the film and a drop or two of the reagent insinuated underneath it.

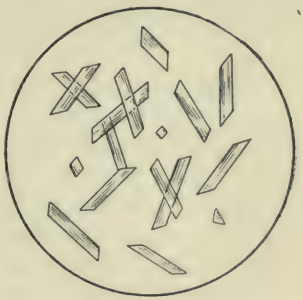


Fig. 20.
Teichmann's Iodo-Hæmatin Crystals.

Over a minute spirit flame the slide is held till the fluid under the cover-slip boils. Care must be taken to replace the reagent evaporated from time to time till boiling has been carried out for about one minute. On cooling, the slide is examined under the microscope with a high power. Black prisms of iodo-hæmatin crystals are seen, if blood be present.

If a negative result be obtained by this test, the suspected material should be subjected to the Guaiac or Aloin reactions, as, from time to time, certain substances are apparently found in the gastric content, containing occult blood, which inhibit the crystalline formation. Otherwise the test is very delicate.

(IV) **Benzidine Test.**—I have placed this test last, although in almost all the text books on clinical medicine and laboratory practice it is placed first, and is often the only one given. The truth is, it is far too delicate—so delicate indeed that it almost always gives a reaction of some kind which may erroneously be regarded as positive when no blood is present.

If the Benzidine test be used, the technique is similar to that employed for the Guaiac and Aloin tests.

The method originally described by Schlesinger and Holst is the one most commonly used :—

(1) In the first test tube dissolve a large pinch of Benzidine in 2 c.c. of glacial acetic acid, so as to make a super-saturated solution.

(2) In a second test tube place a small piece of the prepared fæces (a piece about the size of a pea) with 2 c.c. of distilled water. Boil and set aside to cool.

(3) In third test tube place 10 c.c. of the Benzidine solution, and on top of it a few drops of the fæcal solution, with 20 drops of reliable peroxide of hydrogen.

The development of a distinctly blue colour is a positive reaction.

I think it preferable to carry out the test in a porcelain saucer. A few drops of the Benzidine and peroxide solutions are distributed over the surface of the saucer and a drop or two of the fæcal solution is allowed to flow over this. The character of the blue colour can be very distinctly gauged against the white porcelain.

A pronounced blue colouration may be considered as unequivocally positive, but all blue-green or greenish tints must not be accepted as evidence of the presence of occult blood unless verified by the Guaiac or Aloin tests. I am of opinion that many errors of diagnosis have resulted from the employment of this test, and I know of two patients who were operated on for duodenal ulcer on the strength of a positive reaction. At the operations no evidence of ulcer was found.

This test should probably be abandoned or, if employed, should be controlled by another and more reliable one. The presence of enzymes, saliva, gastric and pancreatic juices, ptomaines, etc., in the gastric content and fæces reacts on the Benzidine, and a blue or blue-green colouration is formed even when blood is entirely absent.

The most scrupulous cleanliness of all apparatus and of the hands is essential in carrying out all the tests for occult blood. Otherwise the results are wholly unreliable.

(V) **Spectroscopic Analysis.**—Spectroscopic analysis is not universally applicable to the gastric contents and the fæces for the detection of blood, but under certain conditions there is no more reliable test.

If the gastric contents are quite fresh and the blood unaltered, the filtrate may be examined at once by the spectroscope for the two *absorption bands of oxy-hæmoglobin*. The first band (left) is denser and more sharply demarcated than the second (right).

If the gastric contents have stood for some time the blood is reduced by the action of the acids to hæmatin hydrochloride,

which is not soluble in water, and examination may fail to give the absorption bands even though blood be present.

If green vegetable be present in the stomach contents the separated chlorophyll gives absorption bands almost identical to those produced by hæmatin.

In examining for blood in the motions, even when the solution of fæces is well diluted, the normal pigments, which absorb light, may mask the spectrum of hæmoglobin derivatives.

To avoid these fallacies it is best to preceed in a definite way and to seek the spectrum bands of acid hæmatin, thus :—

(1) To gastric contents add some water and shake well.

(2) Then add a few drops of sulphuric acid ; shake.

(3) Filter off five c.c. into test tube. Add the same quantity of ether, and shake well to extract acid hæmatin.

(4) Set aside till ether rises to surface ; pipette it off.

By means of a hand spectroscope examine, by transmitted light, a layer one to two c.c. in thickness of the ethereal extract. If blood be present, the characteristic hæmatin bands, in acid solution, will be seen. (For the different absorption bands of blood see diagram.)

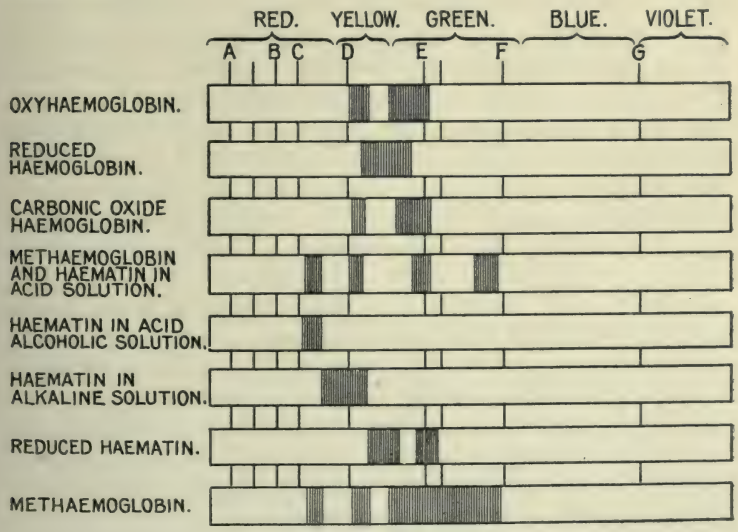


Fig. 21

Spectra of Oxyhæmoglobin and various Derivatives.

C.—The Soluble Ferments.

Pepsinogen and Pepsin.—As for albumin in the urine, so for pepsin in the gastric contents, numerous tests have been employed.

It is generally accepted that the ferments of the stomach are excreted as proenzymes, and that these are activated by the hydrochloric acid present. Lactic and other organic acids may replace hydrochloric acid, but never so efficiently. The amount of pepsin formed from pepsinogen is largely determined by the amount of hydrochloric acid present.

The acid albumins formed in the stomach by the action of hydrochloric acid are rapidly changed by the pepsin into propeptones and peptones.

The determination of the amount and activity of pepsinogen or pepsin present is often necessary and valuable in estimating the functional activity of gastric digestion.

It is rare indeed to find the enzymes totally absent except in cases of very complete atrophy of the mucous membrane. These cases constitute the true achylias.

In many cases of achlorhydria we may find serious defect in the enzyme, although this is not always the case. It may be that, on the addition of the proper proportion of hydrochloric acid to the achlorhydric gastric filtrate, it becomes endowed with the power of converting albumin into soluble peptone. Thus in all cases where we would wish to estimate the presence of a sufficient and active peptic digestion the discovery of an adequate amount of propeptones and peptones in the filtrate, after a peptone-free meal, is proof that the gastric digestive activity is intact.

For the practitioner the easiest test of the presence of pepsin is the indirect one for the detection of *propeptone* and *peptone*, thus :—

(1) To five c.c. of well-filtered gastric content in test tube add an equal quantity of Esbach's reagent, mix, set aside, and note the amount of precipitate of propeptone formed. A rough but useful estimate can thus be made of the presence and activity of pepsin without any incubator or elaborate technique. An Esbach tube may be used for the purpose.

Of the more direct tests the following may be recommended for those who have laboratory facilities :—

(2) If a small slice, weighing about fifteen grains, of the white of hard-boiled egg be placed in a test tube with about five c.c. of

gastric filtrate, and put into the incubator, the action of the pepsin will completely dissolve the egg albumin in from two to four hours.

The same test may be carried out by means of fibrin, complete digestion taking place usually in half the time. The fibrin may previously have been stained in picro-carmine. As digestion goes on the peptic fluid will increasingly become red, from the liberation of the dye, and give an estimate of the rapidity and completeness of digestion.

Peptone can also readily be detected by filtering out the propeptone thrown down by a brine solution and adding to 2 c.c. of the filtrate a sufficient amount of sodium hydrate solution to make it strongly alkaline. On the further addition to this alkaline solution of a few drops of a one to two per cent. solution of copper sulphate, a purple-red or violet-red colour will result in the presence of peptones (biuret reaction).

It should be noted that the activity of the digestive ferments, in vitro, will give, in all probability, a lower reading than would be obtained in vivo, where other adjuvant factors come into play.

(3) **Mett's Test.**—Capillary glass tubes of one to two m.m. calibre are filled with egg albumin. Care is taken to introduce no air. The tubes, after filling, are plugged at each end with dough or new bread, and put into warm water, which is quickly boiled and kept boiling for about five minutes. On removal from the water they are at once hermetically sealed with paraffin and set aside for use.

When a gastric filtrate has to be tested for pepsin, two portions of the capillary tube, 3 m.m. long, are cut off by means of file, and placed for twenty-four hours in the undiluted digestive fluid in incubator. The digestive activity of the gastric juice is estimated by the number of millimetres of albumin removed from the ends of the tube. This is measured by the aid of a pair of graduated millimetre callipers with magnifying glass. The standard unit of measure is the amount of pepsin in a normal gastric juice, 0.18 per cent. HCl, capable of digesting 0.1 m.m. of albumin. The pepsin varies as the square of the length of the digested portion of albumin. Thus two m.m. equal four parts and three m.m. equal nine parts pepsin. The normal gastric juice digests out from five to twelve m.m. of albumin.

(4) **Ricin Test** (Jacoby-Solms).—Make a one per cent. solution of ricin in five per cent. solution of sodium chloride. This solution is cloudy, but it clears on the addition of a varying proportion of gastric filtrate.

The amount of gastric juice required to dissolve the ricin deposit estimates the amount of pepsin present. The technique is as follows :—

(*a*) Take five test tubes and to each add two c.c. of ricin and sodium chloride mixture.

(*b*) Add to each test tube 0.5 c.c. of decinormal hydrochloric acid.

(*c*) To the first four tubes add one c.c. of filtered stomach juice, extracted after Ewald's breakfast, diluted in water in these proportions—1/1000, 1/500, 1/200, and 1/100. To the fifth add one c.c. distilled water alone.

(*d*) Incubate the tubes at 37° C. for three hours.

(*e*) Note which dilution has sufficed to clarify the solution—*i.e.*, which will cause the ricin deposit to disappear.

The amount of gastric filtrate required to clarify two c.c. of ricin solution is *one pepsin unit*. Normally 1 : 100 is sufficiently clarified =100 pepsin units.

(5) **The Edistin Test** (Fuld's Reaction).—This test is very similar to the Ricin one. It is dependent on the fact that Edistin is precipitated by sodium chloride, but Edistan, the pepsin digestive product of Edistin, is not thrown down by sodium chloride solution.

Technique.—(*a*) Take six test tubes and add to each 0.1 c.c., 0.16 c.c., 0.25 c.c., 0.4 c.c., 0.64 c.c., and 0.1. cc. of stomach filtrate diluted 1 : 10 or 1 : 20.

(*b*) Prepare the Edistin solution thus :—Dissolve 0.2 gramme slowly in 200 c.c. of hydrochloric acid solution of 30 acidity—that is, to 60 c.c. of decinormal hydrochloric acid add 140 c.c. of distilled water.

(*c*) To each tube add two c.c. of this solution.

(*d*) Add six drops to each test tube of a saturated solution of sodium chloride.

Normally there is no precipitate at 0.4. When the activity of the pepsin is above or below the normal, we can estimate its amount by noting at which dilution a precipitate appears. In the dilution in which no precipitate has formed, complete digestion has taken place.

If the stomach juice has been diluted 1 : 20 we have this formula if the precipitate is obtained at 0.4 c.c. :—

$$\frac{0.4}{20\times2} = \frac{0.4}{40} = \frac{1}{100} \quad \text{Pepsin}=100.$$

If precipitate appears at 0.25, we have :—

$$\frac{0.25}{20\times2} = \frac{0.25}{40} = \frac{1}{160} \quad \text{Pepsin}=160.$$

Rennet.—It is only in cases of achylia or achlorhydria that it is important to seek for the presence of rennet.

The test for the combined acid should be carried out simultaneously to ascertain if there be any gastric digestion at all.

It may be remarked that the presence of this ferment, or its pro-ferment, has been denied, and it is by some asserted that it is nothing more or less than pepsin itself. It is, however, generally assumed, and with reason, that it has a separate existence, for it can be demonstrated to be present when pepsin is entirely absent.

It is quite unnecessary to test for rennet in an otherwise normal gastric juice for the milk-curdling power of the gastric filtrate bears a very close relation to the peptic efficiency.

Tests.—I.—(*a*) Place in a test tube ten c.c. of fresh milk.

(*b*) Add five c.c. of gastric filtrate, which has been neutralised by decinormal sodium hydrate solution. Shake.

(*c*) Immerse tube in water at 37° C. for ten minutes.

Normally coagulation of the milk will have occurred in this time. If no reaction at all occurs at the end of thirty minutes, rennet is absent.

II. *Boas' Test.*—This test is for cases in which free hydrochloric acid is absent from the gastric filtrate.

(*a*) Take six test tubes, and to each add five c.c. of the following dilutions, in distilled water, of gastric juice—1 : 10, 1 : 20, 1 : 40 1 : 80, 1 : 160, 1 : 320.

(*b*) Add to each tube five c.c. of milk.

(*c*) Add 2.5 c.c. of a one per cent. solution of calcium chloride to each test tube.

The tubes are then placed in an incubator and inspected at intervals, and the lowest degree of dilution at which coagulation of the milk takes place is observed.

Normally hydrochloric acid activates the chymosinogen producing chymosin or rennet, but when it is absent the lime salt replaces it.

If rennet be present in normal amount, coagulation takes place in the first five tubes after half an hour's incubation, and in the last only a loose flocculent precipitate or coagulum is formed.

If rennet be deficient, the coagulum will only be found, in varying degrees, in the first three tubes. When coagulation is confined to the first tube, rennet is absent, for the calcium solution alone will produce that without the presence of rennet.

D.—Examination of Test Meal.

Microscopic Features of the Test Meal.—Before filtration it should be further examined microscopically so as to ascertain the amount of digestion it has undergone, and what, if any, organisms, cells, or pieces of tissue may be present.

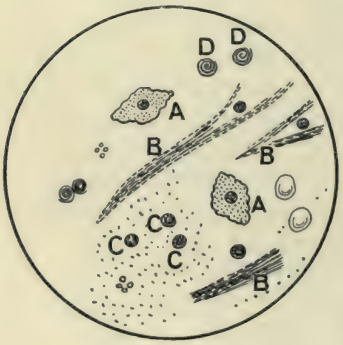


Fig. 22.

Objects normally found in Gastric Contents.

A. Epithelial cells. B. Mucus.
C. Nuclei. D. Jaworski's spirals.

In all cases certain elements intrinsic to the meal and gastric secretion may be present—epithelial cells, starch granules, nuclei, mucus, and of course micro-organisms. Jaworski has indicated the presence of spiral snail-like structures when hyperchlorhydria exists.

No investigation of the vomitus or gastric content would be complete without a microscopic examination. It may sometimes be diagnostic, but it should never be relied upon alone.

The contents of the stomach should be examined both during fasting and during functional activity.

From the empty stomach, after a period of fast, can be extracted a small, but varying, amount of gastric juice. Usually from five to twenty c.c. can be obtained from the normal viscus. Larger amounts are probably pathologic.

To eliminate fallacies the stomach should be thoroughly washed out, with sterile water, just before the test supper is given. Next morning the contents are extracted from the fasting stomach to determine the intactness of the motor function. By following

the ordinary routine by giving a test breakfast in the morning, after the supper on the previous evening, the same information can be elicited.

More important is the examination of the fasting stomach, after thorough washing out the previous evening, for the presence of gastrosuccorrhœa or super-secretion and hyperchlorhydria. We may find that extraction yields an abnormally large amount of highly acid gastric juice.

Duodenal contents (bile and pancreatic juice) may also be found in the empty stomach. If these are obtained in large quantities, obstruction of the lower part of the duodenum may be suspected.

The empty stomach may contain mucus, epithelial cells, red and white blood corpuscles, nuclei, and granular material. Certain organisms and fungi may be found in pathological conditions. Cohnheim considers that free nuclei of the leucocytes is proof of a good peptic digestion.

When excessive mucus is intermingled with a great abundance of columnar epithelium it indicates a common origin from the gastric-mucosa.

If the test breakfast has to be examined microscopically, it is well to wash the stomach thoroughly out the night before, immediately before the test supper of currants. In this way we know that abnormal ingredients found in the extracted breakfast must have been formed during the night. A microscopic examination, however, of the spontaneously vomited material should not be overlooked, for, after lavage, certain organisms may be absent in the extracted test meal that can readily be found in the vomitus.

Method of Examination.—Several films should be prepared by placing a drop of the fresh, mixed contents on several slides, applying cover-slips, and examining at once by low and high powers.

Another set of slides, each also with a drop of gastric contents, should be treated with a very weak solution of iodine insinuated under the cover-slips, and similarly examined.

Of the organisms which may be present the following are of importance :—

Leptothrix Buccalis.—Any of the organisms commonly found in the mouth may be swallowed with the saliva and detected in the gastric contents.

Infusoria. *Trichomonas Hominis* and the *Megastoma Entericum* developing in cases of achylia are, according to Cohnheim, diagnostic of carcinoma of the stomach without impairment of its motility—that is, without retention. Their detection may therefore aid the differential diagnosis of achylia simplex from malignant achylia.

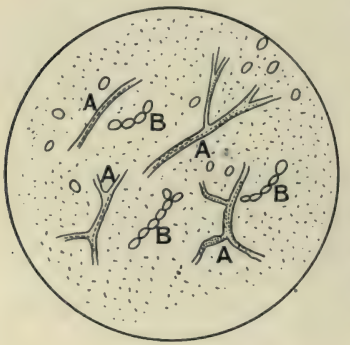


FIG. 23.
A. LEPTOTHRIX BUCCALIS AND B. YEAST CELLS.

Yeast occurs in fermentation associated with simple and malignant stenosis. In simple stenosis, with retention, the yeast cells are usually discrete, but in malignant disease they are apt to form in chains.

Sarcinæ. — These are found only when free hydrochloric acid is present. Thus, when detected in quantity in a test meal or vomitus, their presence strongly contra-indicates carcinoma, and rather favours retention from simple pyloric stenosis. This rule

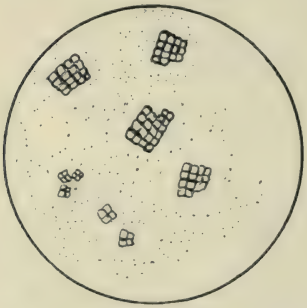


FIG. 24.
SARCINÆ VENTRICULI
(Simple pyloric stenosis).

FIG. 25.
BOAS-OPPLER BACILLI AND YEAST CELLS.
A. Boas-Oppler bacilli. B. Yeast cells in chains (more characteristic of carcinoma). C. Yeast cells discrete.

is not invariable, because occasionally a considerable amount of free hydrochloric acid may persist in advanced gastric carcinoma even with large metastatic development in the liver and elsewhere.

Oppler-Boas Bacilli.—These large non-motile bacilli are not, as is asserted by some writers, absolutely pathognomic of gastric carcinoma. Wherever there is great stagnation with defect of HCl in the gastric contents, they are likely to be found. They certainly occur in about 80 per cent. of all cases of carcinoma, and their presence indicates lactic acid formation.

The organisms occur as large rods very slightly swollen at each end, often lying end-to-end in chain-like series. But they are just as often discrete, and frequently bent or elbowed. When this bent type is abundant it has been said they are more characteristic of carcinoma. I have not been able to verify this.

They stain brown with Gram's solution, and this differentiates them from the leptothrix, which resembles them, which stains blue with this reagent. For their recognition they require no staining, but if it is desired to have them sharply differentiated from the detritus with which they are mixed, a little carbol-fuchsin or methyline blue will accomplish this.

The growth of these organisms is inhibited by an abundance of free hydrochloric acid. When they are associated with large quantities of lactic acid and pronounced constitutional disturbance they are very suggestive of gastric carcinoma.

Kaufmann has shown that the lactic bacillus may act on certain sugars and form lactic acid.

Moulds are not common in the gastric contents. They are found as mycelia and spores. That most frequently met with appears to be the common blue mould. The oidium albicans is also sometimes present. These fungi occur in spite of the presence of an adequate amount of hydrochloric acid, and their occurrence is favoured by a defective gastric motility.

Various parasites may appear in the test meal, such as ascaris lumbricoides and the oxyuris vermicularis.

It will be obvious that the microscopic characters of a test meal will largely depend on the ingredients of which it is composed Thus the returned test dinner will differ materially from the breakfast. Often valuable information can be obtained from this examination, and it ought to be undertaken in every complete investigation of a digestive derangement.

A drop or two of the test meal, picked up from the bottom of a conical glass in which it has stood, should be placed on a microscopic slide, and covered with cover-slip. It should be examined both

by a low and high power lens, with and without the addition of a weak iodine solution.

The remnants of food to be sought for and recognised are chiefly starch granules and meat fibres. The amount of alteration in the appearance of the remnants will depend on the digestive activity of the stomach.

Meat fibres, showing little change, will be readily detected in cases of failure of digestion. They are usually a reddish brown in colour, and show fine longitudinal and transverse striation. Where peptic digestion is active, the striation is likely to disappear, and the meat fibres are then only recognisable by their translucent reddish appearance.

Starch granules always form a prominent feature in a mixed meal and in the test breakfast. They should not be confused with leucocytes. The amount of digestion of starch in the stomach may roughly be said to be in inverse ratio to the acidity. An excellent estimate of this can therefore be formed from the microscopic examination. The starch granule is kidney or spherical-shaped, of various sizes, and somewhat granular or striated. The amount of digestion it has undergone will greatly modify its appearance. The number of starch granules persisting may easily be detected by the addition of a drop of a very weak solution of iodine insinuated under the cover-slip.

If *amyl-dyspepsia* be present, the field will stain almost entirely blue from the unaltered starch granules. Or the granules may show that partial digestion has gone on, and many of them are then burgundy-coloured (erythrodextrin), while others, whose digestion is nearing completion, are unstained by the iodine (achroödextrin).

Vegetable cells of many kinds are also seen, especially after a test dinner, and they are larger than the ordinary starch cells, and sometimes show a greenish tint from the chlorophyll they contain.

Fat drops are recognised by their highly refractive characters.

CHAPTER XIII.

EXAMINATION OF THE FÆCES.

In many cases of gastric disorder an examination of the stools may be required as an aid to diagnosis.

The macroscopic, microscopic, bacteriological, and chemical examination must be undertaken if certain information is to be gained.

The various tests for the detection of blood in the gastric contents are equally suitable for its detection in the fæces.

There is no doubt that the routine examination of the fæces, in digestive derangements, is too much neglected and diagnostic information lost that might otherwise be available. A properly conducted examination of the stool affords information as to certain gastric, pancreatic, and intestinal disorders.

Sometimes the fæces to be examined should be prepared by a known and special diet as that of Schmidt or Gaultier. Usually there is an advantage in giving an "intestinal test meal," although information can be elicited from the examination of the stool on almost any ordinary mixed diet. There are different ways of administering the test diets. One selected meal will suffice if its remnants can be demarcated from the rest of the excreta. By administering, immediately before and after the test meal, either five grains of carmine or twenty grains of powdered charcoal, in cachet, the intestinal remains of the meal will be sharply differentiated from the fecal mass generally by its red or black colour. From the time of ingestion to evacuation per rectum a period of from twenty-four to thirty-six hours is usually occupied.

The Schmidt and Gaultier Diets.—It will be observed that both contain all the ingredients normally composing a mixed meal.

The Schmidt Diet.

Breakfast.—0.5 litre milk, or the same amount of cocoa, prepared with milk, is given along with fifty grams of zwieback (rusk).

Forenoon.—0.5 litre of oatmeal gruel made from oat flour with 10 grams of butter and one egg is given about 11 a.m.

Noon Dinner.—125 grams of lean raw minced beef, lightly cooked, with a large helping of potato purée, with a glass of milk, and some butter, forms the dinner.

Afternoon.—Repeat the morning meal.

Evening.—Repeat the forenoon meal.

This diet is given for three entire days, and the stool passed on the fourth day is selected for examination. By this method there is greater certainty that the stool will really contain the diet given.*

GAULTIER'S DIET.

White bread	100 grams.
Lean beef	60 ,,
Butter	20/30 ,,
Milk	300/500 ,,
Potatoes	100 ,,

The beef is grilled and some sauce made from the butter poured over it. The potatoes are weighed after boiling, and mashed with some of the milk and butter. The remainder of the butter is eaten with the bread along with the milk. The demarcation of this meal in the evacuated fæces is made by means of carmine.

The routine examination of the stool should be made as follows :—

1. **Macroscopic Appearance of the Fæces.**—(*a*) *The quantity* passed, by a man of average weight, on an ordinary diet varies considerably. A great deal depends on the prevailing character of the aliments forming the dietary. If much vegetable material is partaken of, the stool will be correspondingly bulky, owing to the large amount of insoluble waste residuum. On the contrary a small stool will be passed on a concentrated albuminous diet which leaves little waste behind. In the latter condition an evacuation may only take place every second day, due to the lack of stimulation of the bowel by an insufficient detritus. This should not be confused with true constipation.

By weight the normal evacuation varies from four to seven ounces.

A normal stool contains an enormous number of bacteria, many hundred billions, which account for about one-third of its weight,

* The diet given here is somewhat modified from that prescribed by Schmidt as being more suited to the English palate.

undigested food, insoluble residue, epithelial cells, salts and enzymes In addition to these normal there may be many varied and abnormal constituents.

(*b*) *Colour of the Stool.*—The colour depends to a considerable extent on the character of the diet. If milk foods alone are taken, the fæces are normally pale, as is seen in the mustard-coloured stools of infants during the period of lactation. They are darker in proportion to the amount of meat taken or from the ingestion of colouring agents, such as strong coffee or chocolate. The presence of a sufficient amount of urobilin is essential to the normal brown colour of the motion on a mixed diet. In obstructive jaundice the stools are pipe-clay coloured, and in certain cases of pancreatitis the stools may be abnormally pale, especially if fat be present in abundance, when they will also have a greasy appearance.

Many drugs change the colour of the stools. They are more or less blackened by charcoal, iron, and bismuth. A little experience can often determine to which of these drugs the discoloration is due. Podophyllum and calomel produce stools of a greenish tint from an abundance of biliverdine. Blackberries, black currants, and other dark fruits may correspondingly stain the fæces. In recording the clinical history of certain gastro-intestinal diseases the presence of melæna must not be accepted unless these fallacies can be eliminated.

Blood which is macroscopic in the stools may have various appearances. Red blood passed per rectum only during defæcation, and in small quantities, can definitely be recognised as having a local origin from piles, fissures, ulceration, or cancer of the rectum. In such cases the blood will not be intermixed with the motion, but will usually lie on the surface of the fæcal mass.

Red blood, passed in large volume, partly mixed with altered blood, may have its origin high up in the tract. For example, in copious bleeding from a duodenal ulcer the entire gut may be flooded, and the blood be passed per rectum before it has been long enough in the bowel to be much altered by the digestive fluids and intestinal gases.

Malæna, or the black stools of hæmorrhage, is characteristic of a bleeding from a point high up in the digestive tract. Moreover, malæna indicates that the hæmorrhage has not been of the most copious type, and that the blood has flowed slowly enough to lie in the bowel sufficiently long to undergo the darkening change. It is

of most frequent occurrence in gastric, pyloric, and duodenal ulceration. But it occurs in malignant disease of the stomach, in cirrhosis of the liver, and it may be seen in certain blood diseases, where there is a proclivity to hæmorrhage from the mucous membranes.

Blood may be present in considerable quantity in the stools, as in the gastric content, without its presence being suspected (occult blood). It is essential, therefore, to test all stools for its presence in diseases where its detection would be a factor in the diagnosis (see tests for Occult Blood).

(*c*) *Odour.*—This depends very largely on the diet. The more nitrogenous food that is taken the more likely is the stool to be offensive. In pathological conditions, especially in ulceration of the intestine and malignant disease of the stomach, the fæces are often foul-smelling.

The normal odour is due to skatol and indol, which give the stool a sufficiently characteristic smell.

Sulphuretted hydrogen may be excessive and impart its odour to the stools, and often, when acid fermentation is present, the stools have a peculiarly acrid sour smell, and they are always very offensive in obstructive jaundice, pancreatitis, and in most of the diseases associated with intestinal ulceration, enteric fever, tuberculosis, cancer and specific disease.

(*d*) *Consistency of the Motion.*—Normally the first part of the stool should be formed and round, while the later portion is pultaceous

When constipation is extreme the stool is greatly desiccated is voided with difficulty, and the mass is composed of discrete more or less round portions, loosely welded together (scybalæ) These scybalous masses may be passed separately, and are frequently covered with a coating of mucus, produced by the irritation of the hardened masses from too long contact with the mucous membrane. (See chapter on Constipation.)

From a scybalous mass, which becomes encrusted with organic salts, we have the *enterolith.* Enteroliths may be large enough to give rise to symptoms of obstruction and, at times, are responsible for inflammation of the appendix.

Intestinal sand appears to occur mostly in neurotic subjects. Most commonly it is a pseudo-condition. True intestinal sand is of rare occurrence and is composed of carbonates and phosphates of calcium and magnesium mixed with organic material. The

significance and pathogenesis of this condition have not so far been established.

Diarrhœa is present in almost all forms, at varying intervals, of gastro-enteritis, and in all ulcerations of the bowel. The exact consistence of the stool in these conditions is at times informing. (See chapter on Diarrhœa.)

It is not infrequent to find the liquid stool quite frothy from the presence of active fermentation.

Gall Stones.—If these are being sought for, the motion must be broken up and passed through a sieve after being dissolved in water. The fæces should be so examined for several days after a suspected attack of biliary colic.

(*e*) *Undigested food,* mostly vegetable in character, can be readily seen by the naked eye, but, if a healthy intestinal digestion be present, no recognisable meat fibres should be seen.

Large quantities of undigested starch are characteristic of catarrh of the small bowel. The presence of undigested starch can be easily ascertained microscopically by staining a film of the liquified fæces with a solution of iodine.

(*f*) *Mucus.*—Normally a small amount of mucus will be found in most loose stools, and almost always in the evacuation produced by a large washing-out enema.

In health the mucus which is abundantly present in the colon is not seen in the motion as it adheres to the mucous membrane: but when the peristalsis is greatly stimulated, by purgation or enemata, proportionate quantities are passed with the fæces. As a rule it is not intimately mixed, and is unorganised, resembling white of egg, or tinged slightly yellow with bile pigment.

Mucus which appears in the wash-out, after the Plombière treatment, in only moderate quantities is not, as is often erroneously stated, due to a colitis, but may be quite normal. Mucus passed spontaneously, however, in any quantity and persistently, is always abnormal.

The less tinged with bile is the mucus, the more likely is it that it has taken origin in the colon.

It should always be ascertained whether mucus is voided only during defæcation or whether it escapes from the rectum in the intervals. When it is passed independently of defæcation it probably comes from the lower part of the rectum, and may indicate such conditions as piles, polypi, or tumour growth.

Evacuations almost entirely composed of jelly-like mucus, with or without blood tinging, are rather typical of proctitis, and are frequently seen during, or as a sequel to, dysentery.

Mucus which is organised into shreds, membranes or casts, is passed in the so-called membranous colitis. But it is rare that membranes or casts are entirely free from an admixture with glary unorganised mucus.

The mucus should be examined microscopically for accompanying cells. If round or epithelial cells are found intermixed in quantity there is a catarrh of the colon present. The mucus may also require to be examined bacteriologically.

(*g*) *Pus.*—It should be noted that small quantities of pus from the upper tract cannot be detected microscopically, as they are so intimately mixed with the fæces; but recognisable pus passed per rectum is proof of local ulceration or the presence of an abscess cavity, which has perforated and is discharging into the gut. Sometimes the pus may be mixed with blood, or may contain fragments of tissue. Examination of these may give diagnostic information.

(2) **Chemical Examination of the Fæces.**—*Reaction of Fæces.*—After a Schmidt diet, a small portion of the stool, if solid, should be placed in a small mortar and triturated with a sufficient amount of distilled water to reduce it to a creamy consistency. If the stool be already sufficiently liquid it requires no such preparation.

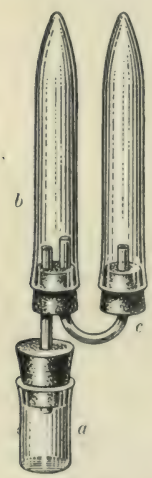


Fig. 26.
Strassburger's and Schmidt's Fermentation Tube.

One end of a small piece of red and blue litmus paper should then be floated on to the surface of the liquified fæces, care being taken to prevent the upper surface of the paper from being immersed, and the reaction ascertained.

The healthy stools should be neutral, or only feebly acid or alkaline in reaction. If the stool be strongly acid, carbo-hydrate fermentation may be assumed. The acidity can often be recognised by the odour due to the presence of fatty or lactic acid. If strongly alkaline, fermentation of albumin is present. The alkalinity is due to ammoniacal decomposition, and it can be detected by the smell, which is extremely offensive.

These fermentations can be fairly accurately gauged by the test apparatus of Schmidt and Strassburger.

Five grams of fresh solid mixed stool (more if it be liquid) are thoroughly triturated with distilled water. The reaction to litmus is then observed, and the fæces, thus prepared, are placed in the lower chamber (*a*) of the apparatus, which is then filled to the top and tightly stoppered so as to exclude all air. Two test tubes, inverted, and fitting tightly into perforated corks, through which pass glass tubes, are then placed in position. Tube (*b*) is then completely filled with water, while tube (*c*) remains empty. The whole apparatus is incubated for twenty-four hours at 37° C. As fermentation goes on the gas formed in (*a*) passes to (*b*) and forces the water out proportionately into (*c*). In the healthy fæces there is practically no fermentation, and therefore no gas-production in the twenty-four hours. If water be forced into (*c*) in an amount equal to one-third of its capacity, it is always pathologic. A small amount of fermentation may be regarded as negligible. The fæces should, when the experiment is completed, be again tested for the reaction. If great or increased acidity accompanies the gas-formation, then we have carbo-hydrate fermentation. The gas collected in tube (*b*) will have the odour of butyric acid, and the fæces will have a higher or brighter colour. If alkalinity be great, or increased from the initial reaction, the gas-formation is due to albuminous putrefaction, and the gas which has gathered in tube (*b*) will have a putrefactive odour. The fæces will also have darkened in colour.

Bile pigment, unaltered, is not found in the healthy fæces. Urobilin, which colours the stools brown, is a normal ingredient. The nitric acid test and the play of colour is not accurate. A watery solution should be made of the fæces by trituration in a mortar. It should be filtered several times till perfectly clear, and tested for bile pigment in the same way as for its detection in urine. About five c.c. of the filtered clear solution of fæces is placed in one tube, and a very weak alcoholic solution of iodine (brandy coloured) run on to the top with care to prevent intermingling of the fluids. Bile pigment is present if a green ring forms at the junction of the two fluids.

Urobilin and bilirubin are easy of detection by taking a small portion of the solid fæces in a glass beaker and covering it with a watery solution of mercuric chloride and placing it for a short

time in the incubator. A reddish-pink colour on the surface of the fæces indicates the presence of urobilin, a green colour of bilirubin.

Urobilin is a normal constituent of the fæces, and its absence is pathologic, and often indicates obstruction of the biliary flow. The presence of bilirubin, on the contrary, is pathologic.

Fat in the Fæces.—The normal fæces may contain small amounts of fat. It is most commonly seen as fatty acid, in spicules, in rosette formation. These crystals may be converted into fat by insinuating under the cover-slip a few drops of sulphuric acid

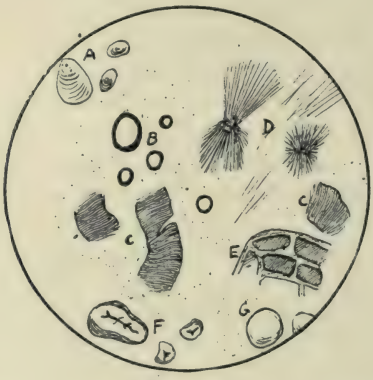


Fig. 27.

A. Potato starch. B. Fat globules. C. Muscle fibres. D. Fat crystals. E. Vegetable fibre. F. Vegetable starch. G. White bread starch.

and gently heating. Or the fat may occur as discrete droplets, easily recognised by their high refractivity. In the absence of bile, in obstructive jaundice, the fat is usually abundant, and is found in drops and crystals. Excessive fat may also be seen in the fæces in any condition where its absorption is interfered with, as in intestinal catarrh and in certain chronic tuberculous conditions of the intestine and peritoneum.

Apart from these conditions the presence of abundant fat in the stools is always pathologic, and for the most part is met with in various forms of pancreatitis. *Steatorrhœa* may be recognised by the naked eye, but it should always be verified by tests.

Albumin in the Stools.—Albumin should not be found in the fæces of the healthy individual. A watery solution should be made

and filtered till clear. Ten c.c. of this, placed in a test tube and acidulated with a few drops of acetic acid, and the upper portion boiled, will determine the presence or absence of albumin. Its presence is of rare occurrence, apart from pus, but it is sometimes found in enteric fever, chlorosis, and acute enteritis.

Peptone and Propeptone should, when albumin is absent, be sought for. The same watery solution of fæces, as employed for the albumin test, is utilised. Some phospho-tungstic acid is added. To the precipitate which forms a weak solution of sodium hydrate and a few drops of a weak solution of copper sulphate are added. A purple-red coloration (the biuret reaction) is positive of the presence of peptone and propeptone. Peptone is normally absent, but may be found in enteric fever, septic peritonitis, dysenteric and tuberculous ulceration of the intestine.

(3) **Microscopic Examination of the Fæces.**—The microscopic examination of the stools after the Schmidt diet is undertaken for the detection of unaltered meat fibres, connective tissue, and the presence of nuclei. The latter are alone digested by the pancreatic secretion. The under-done meat of the diet will supply these ; but Thymus gland, which has an abundant supply of nuclei, is sometimes given to make their detection more easy. A more certain way is to administer a dose of commercial stained nuclei mixed with sugar of milk. Starch granules are also sought for, and the degree of their digestion noted. A search will also be made for the presence of crystals, parasites, and organisms.

Three slide preparations are made of the prepared triturated stool obtained after a three days' Schmidt diet. The presence of undigested meat and starch in a stool taken without preparation might have no other significance than that more of these substances was eaten than could be digested.

(*a*) A small drop is placed on one slide and covered with slip and examined immediately with a high and low power. The normal appearance of such a film will be fairly homogeneous and contain certain constituents of the aliments forming the diet. Vegetable and meat fibres should be so changed as to have lost their histological appearances. Only the fibro-vascular system of the vegetable substances may remain in outline, while meat fibres occur as homogeneous refractive yellow masses.

If meat fibres, well stained and with visible nuclei, be found in the film, a morbid condition is present.

Starch ought to be completely digested and not found in any quantity in the fæces. Unaltered starch, abundantly present, is always pathologic.

Fat may be seen in small highly refractive droplets, or in the form of fatty acid crystals. In a well-digested stool neither of these forms of fat should be found in any quantity.

Crystalline salts are found normally as calcium carbonate, oxalate of lime, and triple phosphates. An abundant deposit of the latter is characteristic of nitrogenous decomposition, and is associated with an abnormally alkaline reaction. These crystals may, however, be found in small numbers in the normal fæces.

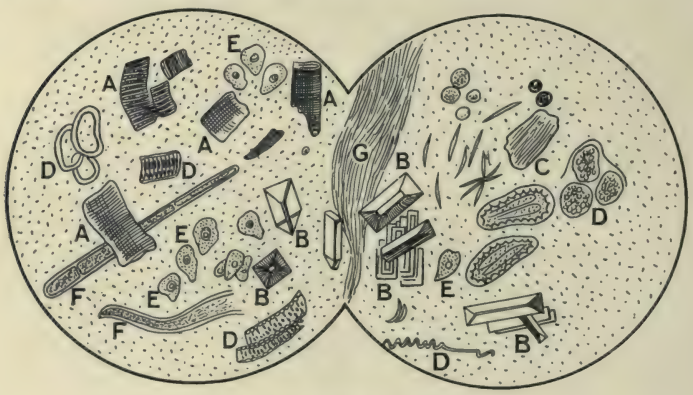


FIG. 28. SLIDE OF FÆCES.

A. Muscle fibres (unaltered). B. Triple phosphates. C. Muscle fibre (partially digested). D. Vegetable fibres and cells. E. Epithelial cells. F. Cuticular substance. G. Connective tissue.

Rhombic crystals of hæmatoidin are met with after gastro-intestinal hæmorrhage, and Charcot-Leyden crystals, slender pointed octahedra, are supposed to indicate the presence of intestinal parasites. More rarely are these found in dysentery, enteric, and phthisis.

Epithelium, mucus, pus, blood, and fragments of tumour tissue may all be got in pathological conditions.

(*b*) A second droplet of the liquid or liquified stool should be placed on another slide, and stained with a strong solution of iodine (Lugol's solution), covered with slip and examined.

Undigested starch cells will show up blue or blue-black (unchanged starch) or burgundy coloured (erythrodextrin). The absence of cells so stained indicates a normal digestion of starch.

Failure of starch in the bowel is comparatively rare. When unaltered starch is found in quantity in the stool, it is probably due to the precipitate passage of the chyme through the intestine.

Vegetable cells, eggs of parasites, and certain organisms will be made more easy of detection by the iodine.

(*c*) On a third slide is placed another droplet and covered with slip. A little glacial acetic acid is insinuated under the cover-slip, and the slide is heated over a tiny spirit flame till the fluid under the slip boils. Care should be taken not to allow the boiling to proceed to desiccation. This may be prevented by the addition of a second or third drop of acetic acid. The slide should be

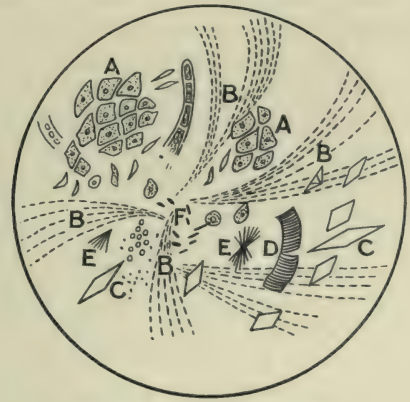


FIG. 29. ABNORMAL FÆCES.
(From a case of chronic intestinal catarrh.)
A. Epithelial cells. B. Mucus. C. Charcot-Leyden crystals.
D. Muscle fibre. E. Fat crystals. F. Bacilli.

examined immediately under the microscope. and while it is cooling crystals of fatty acid may be seen in process of formation.

Micro-organisms.—It has already been noted that these form a large proportion of the weight of the fæces. The most of them, perhaps about 90 per cent., are dead when passed. The flora of the intestine is, under certain conditions, greatly increased. If a drop of the triturate of the stool be placed on a slide and stained with Gram's solution, and then counter-stained with fuchsin, in normal conditions the gram-negative bacillus coli will stain red and the general appearance of the field will be red. When, however, abnormal putrefactive conditions exist, the gram-positive organisms will predominate and give the field generally a blue appearance.

Thus the appearance of the field affords valuable information as to the absence or presence of abnormal putrefactive processes in the intestine.

As a rule there is a direct ratio between the abundance of gram-positive organisms and the amount of nitrogenous material forming the diet. Certain organisms flourish most when the diet is largely nitrogenous, and others when it is principally carbo-hydrate.

Micro-organisms found in the fæces belong to the aerobic or anaerobic group. The anaerobic organisms are associated with the putrefactions in the bowel, and are very important as potentially pathogenic. When present in excessive numbers they are responsible for various auto-intoxications. The more important of these are B. putrificus and paraputrificus and B. aerogenes capsulatus. These organisms are gram-positive.

The B. coli communis, which belongs to the aerobic group, is the organism which predominates, but many coliform organisms are also found.

The lactic acid forming bacilli are credited with inhibiting the action of the putrefactive organisms.

Various specific organisms of enteric, dysentery, and cholera, etc., are to be found in the stools during the course of these diseases. The reader is referred to various works on bacteriology for a complete account of this subject.

A culture prepared from the fæces also affords information as to the character and numbers of the organisms present. Bacteriological examination of the fæces, although from time to time yielding valuable information as to morbid processes, requires a special knowledge and technique which renders this mode of investigation of no great utility to the practitioner.

It is not within the scope of this work to give a detailed account of the parasites met with in the fæces; but as some of these are associated with gastro-intestinal disorders it may be at times necessary to exclude them as possible factors of disturbance.

The common parasites are the Ascaris Lumbricoides (round worm), the Oxyuris Vermicularis (thread worm), the Cestodes (tape worms—Tœnia Solium, T. Mediocanellata and T. Bothriocephalus Latus), Echinocci, Tricephalus Dispar (whip worm) Trichina Spiralis, Trematodes (fluke worms), and Uncinaria Duodenalis (hook worm). The characteristic ova of many of these parasites may also be found in the fæces, and their presence may enable a diagnosis to be made.

CHAPTER XIV.

MOTOR POWER AND ABSORPTIVE CAPACITY OF THE STOMACH.

Having examined the stomach physically, and its contents chemically and microscopically, it may be necessary to obtain, by other methods still, information as to its motor power and absorptive capacity.

Motor Power.—We have already noted in the extracted breakfast whether any remnants of the supper were found and what amount of breakfast was returned. From these data reliable information as to the motility of the stomach may be gathered. Many aberrations of the secretory functions, such as hyper-acidity and achylia, may give rise to no symptoms; but any serious loss of motor function is immediately productive of grave disorder.

If a stomach be syphoned seven hours after a test dinner, such as that of Riegl's,* and no remnants be found in the wash-out, it may be inferred that it is not lacking in motility and that there is no serious stenosis.

On the other hand, it is certain that when remnants of previous meals, sometimes taken twenty-four or forty-eight hours previously, are found on syphonage or lavage, we have some gross interference with the outlet of the stomach.

The loss of gastric motility, resulting in retention, should not be confused with the stasis due to pyloric stenosis. In the latter condition, when the obstruction is recent, the peristalsis of the stomach is usually increased from "compensatory hypertrophy" of the muscular tissues. Compensation may fail later, when we get "atony" added to the stenosis.

Ewald's Motility Test.—This method of ascertaining the gastric motility consists in the administration of fifteen grains of Salol

* One plate of meat broth, a beef steak, potato purée, and a roll of bread.

half an hour before food. At the same time the bladder is emptied. The patient is then instructed to micturate every half hour into specially numbered urine glasses. Each sample is then tested for salicyluric acid with ferric perchloride solution. The burgundy-red or logwood colour which follows the addition of the ferric chloride is proof that the salol, which is decomposed into its constituents (salicylic and carbolic acids) in the alkaline secretions of the intestine, has left the stomach.

In health the reaction is positive in from thirty to seventy minutes. If there be stasis or stenosis, the reaction may be delayed for two or three hours, or indefinitely. All the salicylic acid should have been eliminated from the normal patient in about twenty-four hours. In gastric stasis this limit may be exceeded twice or thrice.

The Iodipin Test.—This drug, also only decomposed in the intestine, has been employed for the same purpose. With the test breakfast twenty grains of iodipin are given. When the stomach begins to empty, and the drug passes into the intestine, free iodine is liberated and quickly absorbed into the circulation and excreted in the saliva. By dipping starch paper, at fixed intervals, into the mixed saliva, and touching the papers with a glass rod which has been dipped into fuming nitric acid, the presence of free iodine is readily detected. The test should be made every quarter of an hour and, in health, should be positive not later than sixty minutes after the administration of the capsule containing the iodipin.

Klemperer's Test.—Wash out the stomach. Introduce 100 c.c. of olive oil before removing the stomach tube. Aspirate the stomach two hours later. Oils are not absorbed by the stomach. If more than forty c.c. can be extracted after two hours, the motor power of the stomach is defective. The larger the return of oil the greater the loss of motility.

By means of X-rays and a full opaque meal (four ounces of bismuth carbonate or barium sulphate in one pint of arrowroot gruel) the motor power of the stomach, the condition of the pylorus, and the rate of emptying may be simultaneously observed, with a considerable degree of accuracy, on the fluorescent screen.

Absorptive Capacity of the Stomach.—The method by which prepared aliments are absorbed from the stomach is not definitely understood. Certain substances appear not to be absorbed from the gastric mucosa. Neither water nor fats are taken up by the gastric mucous membrane, although alcohol, albumin, dextrin,

peptone, and sugar appear to be in small quantities. Water leaves the normal stomach, especially when taken alone without solids, with remarkable rapidity.

Penzoldt and Faber gauge the absorptive capacity of the stomach by the administration of potassium iodide. Three grains of potassium iodide are administered in a gelatine capsule, and this very diffusible substance, when absorptive power of the stomach is normal, should be detected in the urine and the saliva, tested every two minutes, in from six to fifteen minutes. The test is conducted in exactly the same way as above, with iodipin. In delayed absorption there is a corresponding delay in detecting the blue colour or positive reaction. This test is not very reliable.

CHAPTER XV.

DIETETIC PRINCIPLES.

There are certain principles which must guide the physician in selecting a diet for any condition. These are that the diet should be (1) suitable for the special condition which may prevail in the gastro-intestinal tract, and (2) adequate for the needs of the body.

Too often 'diet' is only another name for starvation. Diet should be food and medicine combined—that is, it should be sufficient to supply the requirements of the system in maintaining a proper balance between waste and repair, and exert a useful local action on the morbid digestive condition for which it is prescribed. At the very least the diet advised should never have a harmful influence on the morbid process.

The problem of diet is always a difficult one, for it involves many considerations. It is not merely a question of giving the physiological number of calories of the different essential materials. We have also to ascertain whether a patient suffering from a defective digestive power can utilise these ingredients. The value of any aliment largely depends on its combustibility and the amount of heat which is evolved in the process. Every fuel is not the same. Some will evolve a certain amount of heat with the formation of a certain amount of waste product; others will give a less amount of heat and more waste residue. These principles must be borne in mind in framing a diet.

The larger proportion of the food we eat is employed in generating heat. A very small quantity suffices for the repair of waste in the tissues. Two standards of heat units have been adopted. By a heat unit or 'calorie' is meant the amount of heat required to raise one gramme of water one degree centigrade. This is known as the *small calorie*, while the *large calorie* is the amount of heat required to raise one kilogram one degree centigrade. Each large calorie thus represents 1,000 small calories.

Each aliment or fuel has its own caloric equivalent when it undergoes combustion, and although theoretically we might obtain from one single ingredient a sufficient number of calories to supply the body needs, it will be found practically that better and more economic results are obtained from a mixed fuel, or dietary, than from the single ingredient aliment.

It is believed that a healthy adult man, of average weight, gives off 2,400,000 small calories of heat daily, and we therefore ought, as far as possible, to restore this expenditure by the daily diet. It is well to bear in mind that even in health there is a good deal of our nutriment unutilised which is excreted in the fæces, and that in certain digestive derangements this quantity may be increased. Allowance must therefore be made for it, and a diet prescribed somewhat larger than that supplying the exact physiological requirements.

If we could collect all the ash or oxidation waste products of our diet, we could arrive, with some degree of accuracy, at the amount of nutriment which should be given. But this is not possible, for neither by an examination of the urine nor of the fæces for these oxidation products should we obtain the figures we seek. A considerable proportion of the oxidation products is intangible, at least for clinical purposes, and passes out of the body by the lungs and skin.

As a rule far too much proteid is recommended in the standard dietaries, resulting in the formation of too large an amount of waste products only to tax, sometimes to injure, the organs of elimination.

But whatever standard we adopt, it is imperative that consideration be given to a number of factors—viz., age, body weight, work, climate, and individual peculiarity.

It will be more convenient to work in figures of large calories in framing a dietary.

For an average man of about 140 lbs. in weight, doing an ordinary day's work, 3,000 to 4,000 large calories are required. If a man's work be very strenuous, a larger number of calories should be supplied to recoup for increased tissue waste.

Sometimes the diet built on strictly physiological principles will be found to be insufficient to adequately nourish the patient. If weight be consecutively lost on a diet framed on physiological data, it should be reconsidered, and probably be made more liberal.

Generally speaking, the diet of a healthy man must contain :—

Nitrogenous (albuminoid)	2 to 3 parts.
Fat	3 to 4 parts.
Carbohydrates	5 parts.
The salts essential.	

Nor can the caloric value of any aliment alone be the guide. The following four substances have these caloric values :—

10 grams of	Albumin	41 calories.
,, ,,	Carbohydrates	41 ,,
,, ,,	Fat	93 ,,
,, ,,	Alcohol	70 ,,

But it will be obvious to anyone that the same quantities of these different materials, although producing different amounts of heat, are not to be valued as alimentary substances in proportion to the heat they generate on combustion. We cannot, therefore, construct a diet by supplying only the requisite number of calories.

In estimating the appropriateness of any diet a great deal depends on the capacity to utilise the nourishment given. This differs greatly in the normal, and perhaps more in the abnormal, individual.

A very large amount also depends on the environment of the individual and whether he be at rest or at work. We get a good illustration of this in the life of the chicken. During its period of incubation in the shell it finds ample nourishment for its growth and construction in the contents of the egg—contents formed of different albumins and only a small amount of carbohydrates. But during the period of incubation the chicken is at rest. When it escapes from the shell and begins a life of active movement, it at once seeks and thrives on a totally different form of nourishment, now composed almost entirely of carbohydrate and a relatively small proportion of vegetable albumin in the grains it consumes.

These facts correspond with our scientific knowledge that for the growth and construction of the body and the maintenance of repair, nitrogenous materials are essential, but for the production of heat and energy a large amount of carbohydrate is required. We may also infer from this that the actively moving young child requires a diet richer in carbohydrates than in albuminous material. Proteid material in adequate quantity is, however, essential. It is utilised in the processes which permit of the metabolism of starches as heat producers.

Two types of aliment, each respectively in themselves almost complete foods, are eggs and milk.

Eggs contain :—

Nitrogenous material	12.6
Fatty material	12.1
Non-nitrogenous material	0.5
Salts (inorganic)	1.1

It will thus be seen that eggs present the materials for building up and repairing waste in a unique way. Compared with an equal quantity of beef steak, egg is a superior nutriment, and it is extremely easy of digestion.

The nutritive value of 100 grams of egg is equivalent to 158, and that of 100 grams of beef to 124 calories.

Eggs contain globulin and other highly specialised albumins, and lecethin, and a rich supply of fat is present.

If we add to egg some carbohydrate, in the form of starch, we make of it a most complete food for all the requirements of the body—a food competent to build and repair the tissues as well as to supply energy for work.

Milk contains :—

Albuminoids....	3.5 per cent.
Fats	3.7 ,,
Lactose	4.9 ,,
Salts	0.7 ,,
Water	87.2 ,,

Milk may be regarded as a complete food. While it is generally true that it is an essential in the treatment of all the more acute gastric and intestinal ailments, there may be a few conditions for which milk is quite unsuited and even injurious. It will usually be found that when it appears to disagree in its unaltered form, it may be made to suit almost everyone and almost every condition by treating it in special ways.

If we refer to the tables of the physiological diet showing the amount of albumin, fat, and starch required by the healthy individual, it will be seen that milk contains these essentials almost in their proper proportion.

It would thus be possible to live on milk alone. Like eggs, milk is very easy of digestion. The large proportion of water which it contains makes it, however, a very dilute diet, requiring

to be taken in large quantities to supply the necessary amount of nutrition.

To obtain from milk 2,700 calories, about four litres (seven pints) would have to be consumed daily. Again, milk is too simple an aliment to be alone relied on as a permanent diet. Even though it were taken in the amount necessary to produce the required 3,000 calories per day, a healthy adult could not work and thrive on it for long. These considerations constitute limitations to its use if employed alone as a food.

When milk is used in the treatment of acute digestive derangements, it should be sipped slowly, mixed well in the mouth with the saliva, almost—one might say—chewed. If this is done, there are few cases, other than pyloric stenosis and extreme hyperchlorhydria, with which milk will be found to disagree. By exception we meet with individuals who have an idiosyncrasy to milk, and, give it how we may, it disagrees in one way or another.

Chittenden, in his classical researches in dietetics, insists too strongly on the strict adoption of the physiological principles in diet. Many other factors, however, come into play. The results of individual and age-long racial experience must count for much. It is obvious that the dietary suitable for the European would not prove suitable for the South Sea Islander.

In health, and more particularly in disease, to get the best out of any diet, we must exercise a certain elasticity in selection, in conformity with the habits and experience of the patient.

Even though we frame a dietary which is perfect from the physiological standard, other considerations are of equal importance. Is it palatable, well cooked, and does it adequately maintain nutrition ?

As has already been pointed out in the chapter on the physiology of digestion, the psychic element plays an important part in the process of digestion. A well-cooked, well-served, and appetising meal, eaten in cheerful surroundings, is certain to be better digested and prove more nutritious than a similar repast badly cooked and served in a slovenly fashion.

Psychic juice is promoted by the pleasurable anticipation of an agreeable meal. The secretion is stimulated reflexly by the senses of smell, sight, and taste. That an abundant output of gastric juice may be so formed, without the actual entrance of food into the stomach, can be demonstrated by the '*sham feeding test.*'

This observation may be carried out as follows:—The tube should be passed in the morning to make sure that the stomach is empty. An appetising meal is then chewed for ten minutes. Careful instructions are to be given that none of the food is to be swallowed, but all spat out. During the next ten minutes all saliva secreted is also to be rejected. The stomach tube is again passed and the gastric contents extracted.

From 60 to 90 c.c. of clear fluid, free from food material, is normally obtained, containing amounts of free and total acid comparable to that found in an actual test meal which has been ingested.

These and many other observations show the importance of the psychic element in digestion, and how detrimental it must be to enforce too rigidly and exclusively a physiological diet.

Great care must also be exercised to prevent good food from being rendered unpalatable and reduced in its nutritive value by bad cooking.

The following tables, compiled from Atwater and Wood, Department of Agriculture, U.S.A., and from Koenig, will be helpful in showing the relative nutritive value and composition of some foods commonly in use:—

Composition of some Foods in common use.

I.—*Meat and Game.*

Food.	Proteid per cent.	Fat per cent.	Carbohydrate per cent.	Calories per 100.
Beef (lean)	20.78	1.50	..	99.15
Beef (fat)	17.19	26.38	..	315.81
Veal (lean)	19.84	0.82	..	86.97
Veal (fat)	18.88	7.41	0.07	146.61
Mutton (lean)	17.11	5.77	..	123.81
Mutton (fat)	14.80	36.39	0.05	399.31
Pork (lean)	20.25	6.81	..	146.36
Pork (fat)	14.54	37.34	..	406.88
Sweetbread	22.0	0.4	..	93.92
Chicken (lean)	19.72	1.42	1.27	99.10
Chicken (fat)	18.49	9.34	1.20	167.58
Partridge	25.26	1.43	..	116.85
Pigeon	22.14	1.0	0.76	100.02

II.—*Farm Products.*

Food.	Proteid per cent.	Fat per cent.	Carbo-hydrate per cent.	Calories per 100.
Milk (cow's)	3.41 to 4.3	3.0 to 3.8	3.7 to 4.81	56.41 to 71.93
Cream	3.61	26.75	3.52	276.01
Whey	0.85	0.23	3.03	18.0
Buttermilk	3 to 4	0.93 to 1.3	3 to 4	33.08 to 43.63
Butter	0.5	90	0.5	823.1
Cheese	34.99	11.37	5.40	269.06
Cheese (Cream)	16.28	41.22	1.9	449.54
Eggs (Hen's—whole egg)	12.5	12.1	0.5	165.0
White of Egg	12.67	0.25	..	54.22
Yolk of Egg	16.24	31.75	0.12	355.99

III.—*Cereals and Vegetables.*

Food.	Proteid per cent.	Fat per cent.	Carbo-hydrate per cent.	Calories per 100.
Wheat Flour	8.5	1.25	73	345.78
Oatmeal	12.05	5.26	66.77	338.80
Barley Meal	8.31	0.81	75.19	323
Wheat Bread	6.0	0.75	52	245
Sago	0.5	traces	86.5	356
Rice	5.5	1.5	76	348.10
Beans	19.5	2	52	311.75
Peas	19.5	2	54	319.95
Potatoes	1.5	..	20	88
Cauliflower	2 to 5	0.4	4	35
Carrots	1.04	0.21	6.74	33.85
Spinach	2.49	0.58	4.44	33.67
Asparagus	2	0.3	2.5	20
Brussels Sprouts	4.83	0.41	6.22	49.05

IV.—*Fruits, Nuts, and Sugar.*

Food.	Free Acid %	Proteid per cent.	Fat per cent.	Carbohydrate per cent.	Calories per 100.
Apples	0.82	..	..	7.22	29.6
Apricots	1.16	..	..	4.69	19.22
Grapes	0.79	..	..	14.36	58.87
Peaches	0.92	..	..	7.17	29.39
Pears	0.20	..	..	8.24	33.78
Plums	1.50	..	..	4.68	19.18
Strawberries	0.93	..	0.45	6.78	31.88
Chestnuts	..	5.48	1.37	38.34	192.11
Cane Sugar	..	0.35	..	93.33	382.65
Beet Sugar	..	..	..	99.75	408.97
Honey	..	1.20	..	73.22	305.22

V.—*Soups, Tea, Coffee, etc.*

Food.	Proteid per cent.	Fat per cent.	Carbohydrate per cent.	Calories per 100.
Beef Tea	0.5	0.5	..	6.6
Meat Juice (expressed)	6 to 7	0.5	0.5	31.20
Malt Extract	8 to 10	..	55	258.30
Rice and Milk	8.8	3.5	28.6	182.61
Tea	12.38	..	..	50.75
Coffee	3.12	5.18	..	59.92
Beer	0.5	5.25	0.3	51
Porter	0.7	6	0.3	60

In considering the caloric values of food materials, as shown in the above tables, the digestibility of the various articles must also be noted in framing a dietary.

Recent experimental observation seems to have demonstrated, what for long has been familiar to experience, that certain principles in food are essential to the maintenance of a proper nutrition, growth and development. These principles have been called *vitamins*. The truth is that vitamins are more or less present in all our wholesome foods when in their raw or natural state, while cooking largely destroys them. The absence of vitamins is responsible for "deficiency diseases," such as rickets and many forms of malnutrition. Fresh milk, cream, butter, fats, oils, raw eggs, ripe

uncooked fruits, etc., are all rich in vitamins, and when possible adequate proportions of these should enter into every well-framed dietary.

Fats and oils diminish the secretory and motor functions of the stomach, and hence cause a delay in the evacuation of the chyme.

This table (after Penzholdt) gives approximately the time required by the stomach to empty itself of measured quantities of certain common foods :—

100 to 200 grams of boiled milk 200 grams of plain meat broth 100 grams of soft egg 200 grams of beer	from 1 to 2 hours.
200 grams cocoa and milk 400 grams light wine 400 grams beer 300 to 500 grams boiled milk 100 grams eggs in any form 200 grams boiled fish 75 oysters 150 grams boiled cauliflower 150 grams boiled potato 70 grams light bread (stale)	from 2 to 3 hours.
230 grams spring chicken or partridge (boiled) 190 grams chicken or partridge (broiled) 250 grams raw or cooked beef 160 grams boiled ham 100 grams roast veal 100 grams raw beef steak (minced) 100 grams roast beef 150 grams bread, biscuit, cereals, vegetables, fruit	from 3 to 4 hours.
250 grams fillet (grilled) 250 grams ox tongue 250 grams broiled ham 280 grams roast duck 250 grams goose (roasted) 150 grams lentil purée 200 grams pea purée	from 4 to 5 hours.

This table is limited in the number of food materials, but many similar articles of diet have approximately the same digestibility.

CHAPTER XVI.

RECTAL ALIMENTATION.

There are many gastric diseases in which it is desirable or imperative to keep the stomach at rest, as far as that is possible.

In ulceration, and especially after hæmorrhage, it may be considered unsafe to introduce any food into the stomach. Or when the gastric mucosa is very irritable, as in acute gastritis, and vomiting is persistent and intractable, it is useless and usually contra-indicated to attempt oral administration till the stomach ceases to resent the intrusion of food.

In all forms of obstruction at the outlet, with severe stasis, it serves no good purpose to add a fresh supply of food to that which is fermenting or decomposing in the stomach.

After operation on the stomach, or abdomen generally, it may for a time be advisable or necessary to give nothing by the mouth.

For all these, and perhaps for other conditions, rectal alimentation may require to supplement or replace oral feeding.

Rectal enemata may consist of simple normal saline solution, or certain alimentary substances may be added to make the nutrient enema.

There is a rather widespread belief that so-called *nutrient enemata* are capable of replacing oral alimentation and of maintaining an adequate degree of nutrition.

It should, however, be clearly understood that any form of rectal feeding must be, at best, a poor substitute for oral alimentation. The rectum has little digestive capacity, and fats, albuminous and other nutrient materials, thrown into it in their ordinary forms, instead of nourishing the patient, tend to undergo decomposition and produce auto-intoxication.

The colon readily absorbs water, saline and saccharine materials, and certain albuminous substances which have been specially prepared. After hæmorrhage, when the tissues have been dehydrated, absorption of water takes place with great rapidity.

The more simple rectal enemata can be kept, the more efficient will they be. All the complex formulæ for nutrient enemata, even

when the ingredients are predigested, tend to produce rectal irritation and auto-intoxication. If they apparently serve the purpose for which they are given at the outset of their administration, it is to the normal saline solution that they owe their efficacy.

I have verified to my own satisfaction that acetonæmia may rapidly follow the use of the so-called nutrient enemata, and that the auto-intoxication deepens the longer the enemata are persisted in. When they are entirely stopped, and normal saline with glucose substituted, the acetonæmia quickly subsides. It seems to me, then, that the complex nutrient enema is worse than useless.

The most important part of any nutrient enema is the water with the saline in solution. By its means alone the patient may be tided over the critical post-operative days or be resuscitated from the collapse of hæmorrhage.

When we are dealing with gastric ulcer we ought to remember that complete gastric rest will not be obtained by rectal alimentation. There seems little doubt that each injection evokes some peristalsis throughout the gastro-intestinal tract, and probably some gastric secretion.

Thus, if for any reason rectal alimentation has to replace oral feeding, the best results will be obtained by the rectal injection of warm normal saline solution to which has been added varying proportions of glucose. If other nutrients in addition to the glucose are desired, they should be entirely soluble, non-decomposable, and capable of complete and rapid absorption. Substances which are readily dialysed generally fulfil the requirements.

Thus albuminoids and peptones may enhance the value of the injection. I have found for this purpose Panopeptone useful in doses of from half to two ounces. Liquid Peptinoids, which is a dialysed product, is also an excellent addition where special support is required. It may be added to each injection in from ʒii to ʒiv.

Instead of glucose, extract of malt may be used, or a decoction of crushed raisins in the preparation of the rectal injection.

Certain medicinal substances may conveniently be added when indicated. Alcohol, adrenalin, and pituitrin are useful in preventing syncope in certain cases.

Glucose is rapidly absorbed; it maintains the action of the heart, and has considerable caloric value, which prevents tissue waste and the exhaustion of the carbo-hydrate reserves.

Great care should be exercised in the administration of rectal enemata. It is necessary first to clear and cleanse the rectum by warm saline solution and after a period of rest the injection may be given. The long, soft, well-lubricated rectal tube should be employed, and the warm fluid be allowed to run by gravitation from a glass funnel while the patient lies on his left side with the buttocks well raised. No air must be introduced, or peristalsis may be set up and the rectal injection prematurely returned.

If rectal feeding has to be undertaken longer than a few days, great care is required to prevent rectal irritation. This can best be avoided by skilful technique and the observance of certain points.

The temperature of the enema should be, in the funnel, not less than 104°F., so as to allow for a certain amount of cooling as the fluid passes down the tube. In this way it will reach the rectum about 100°F. Lanoline should be used to lubricate the rectal tube.

If peristalsis seems to be set up before the desired amount of fluid has been introduced, the flow should be temporarily arrested till the desire to evacuate it passes over.

When the tube is withdrawn the anus should be wiped with a solution of borax and boric acid, and a little anæsthetic renaglandin or compound adrenalin ointment applied to the anal orifice. If these procedures are adhered to, it is surprising how long rectal and anal irritation may be postponed.

From ten to twenty fluid ounces of normal saline may be given to an adult every four to six hours, but if the larger quantities are not well tolerated it is advisable to employ a small enema of from three to six ounces, and repeat it more frequently.

Should persistent difficulty be experienced in passing the tube or the fluid into the rectum, some obstruction may be suspected. To determine this further examination with the finger, sphygmoidoscope, or by X-ray investigation may be required. An examination of the abdomen may reveal the nature of the difficulty. I have frequently found marked succussion in the epigastric region from dilatation of the colon above the site of obstruction. The fluid retained in the colon causes such loud succussion that it is supposed to be gastric in origin. It is, however, more hollow in character.

Many formulæ for nutrient enemata will be found in works on dietetics, but I am so convinced that they are more harmful than useful that I refrain from giving any of them in detail.

SECTION II.

ORGANIC DISEASES OF THE STOMACH.

Classification of Diseases and Disorders of the Stomach.—It is almost impossible to frame a logical, scientific, or purely pathological classification of Gastric Diseases.

Organic and functional lesions are inextricably interwoven. A large number of the 'dyspepsias' are named after one of their leading symptoms, such as 'flatulent dyspepsia,' without regard to their pathogenesis. The well-recognised organic lesions can, of course, be classified pathologically—*e.g.*, gastritis, peptic ulcer, and carcinoma.

In describing the commoner diseases of the stomach I have compiled the following classification purely for convenience in dealing with the subject.

I.—Organic Diseases.

(1) *Gastritis*
- Acute
  - Simple.
  - Phlegmonous.
  - Toxic.
  - Infectious.
- Chronic
  - Simple.
  - Acid.
  - Hypertrophic and Sclerosing.
  - Atrophic.

(2) *Peptic Ulcer*
- Acute
- Chronic
  - Gastric.
  - Pyloric.
  - Duodenal.

(3) *Neoplasms*
- Simple
- Malignant
  - Carcinomata.
  - Sarcomata.
  - Granulomata, etc.

(4) *Deformities and Displacements*
- Acquired
  - Pyloric Stenosis.
  - Hour-glass Stomach.
- Congenital
  - Gastroptosis.
  - Pyloric Stenosis.

II.—Functional Diseases.

- (1) *Dynamic Disorders of*
  - Secretion
    - Hyperchlorhydria.
    - Gastrosuccorrhœa.
    - Achylia Gastrica.
    - Heterochylia.
    - Amyxorrhœa Gastrica.
    - Myxorrhœa Gastrica.
  - Motor Function
    - Myasthenia Gastrica.
    - Acute Gastric Dilatation.
    - Chronic Gastric Dilatation.
    - Cardiospasm.
    - Peristaltic Unrest.
    - Aerophagy.
    - Pneumatosis.
    - Merycismus (Rumination).
    - Regurgitation.
- (2) *Sensory Disorders*
  - Neurasthenia Gastrica (Nervous Dyspepsia).
  - Hyperæsthesia Gastrica.
  - Gastralgia Nervosa.
  - Bulimia.
  - Polyphagia.
  - Sitisphobia.
  - Parorexia.
  - Anorexia Nervosa.
- (3) *Symptomatic and Reflex Dyspepsia from Diseases in other Organs.*
  - Cholelithiasis.
  - Cholecystitis.
  - Pancreatitis.
  - Intestinal Disorders.
    - Obstructions.
    - Appendicitis.
    - Constipation.
    - Diarrhœa.
  - Pulmonary Affections.
    - Dyspepsia of Phthisis, etc.
  - Cardiac Affections.
  - Nephritis.
  - Diabetes.
  - Anæmia.

CHAPTER XVII.

GASTRITIS.

It may be well to understand that the term gastritis does not describe a single clinical entity, nor does it do more than indicate a leading feature of a gastric change—viz., inflammation. In the stomach, as elsewhere, we have many causes of inflammation and various types of it. Acute gastritis is frequently called Catarrhal Gastritis, or simply Gastric Catarrh. These different names merely describe a clinical feature of the disease. As the inflammatory change is not confined to one structure alone, several symptoms, from disorder of special tissues, may supervene. Thus in gastritis the mucous glands are disturbed in their function, and excessive mucus is excreted, giving rise to the name of gastric catarrh.

Gastritis may be acute or chronic, and give rise to corresponding histological changes to those, for example, occurring in acute and chronic nephritis. Naturally the chronic varieties are those associated more often with grave structural organic change ending, not infrequently, in complete atrophy of the mucosa (chronic atrophic gastritis).

To the acute variety the names of Acute Dyspepsia and Simple Gastritis have been given.

That produced by the local action of corrosives and irritants is commonly designated acute Toxic Gastritis, while the more rare form associated with suppuration of the stomach is spoken of as Abscess of the Stomach or Phlegmonous Gastritis, and it may be secondary to pathological changes in other organs or a primary disease.

The type associated with the action of organisms is designated Infective, and that excited by fungi, Mycotic Gastritis.

Another type not due to ingestion of toxic substances, but to their excretion through the mucous membrane of the stomach, may be shortly considered here. It may be described as Symptomatic or Eliminative Gastritis. Thus, in acetonæmia, uræmia,

morphine poisoning, etc., the toxic agents are largely eliminated through the gastric mucosa, and give rise, in proportion to their amount and their irritative properties, to signs of acute gastritis. When certain eliminative channels are interfered with, others attempt to act vicariously. A good example of this is seen in the elimination of urinary waste products through the gastric mucous membrane in uræmia. In this condition we often have symptoms of acute gastritis of the eliminative type.

It becomes obvious, then, that gastritis includes a number of affections producing different symptoms, according to the acuteness of the inflammation and the exact tissue in the stomach most involved. At one time only the parenchymatous may be implicated; at another, principally the interstitial tissue ; while at another all the structures may participate in the inflammatory process and become histologically changed.

From the clinical point of view the classifications based on histological changes have little importance. What matters principally is the fact that, not infrequently, in the chronic varieties of gastritis we can have marked change, even complete atrophy with failure of normal secretion, without serious inconvenience to the patient, as long as the pyloric function and the motility of the stomach remain fairly intact. But in the more severe forms of acute gastritis gastric symptoms are always pronounced.

I. Acute Catarrhal Gastritis.

(Acute Dyspepsia : Gastric Catarrh : Simple Gastritis.)

This condition is very common, and constitutes a very large proportion of the acute dyspepsias or indigestions.

Etiological Factors.—Any irritant substance which has been ingested may provoke an attack. Certain foods, bad in themselves or improperly prepared, may give rise to acute gastritis. Excessive quantities of sound food may have a similar effect. Tinned foods, bad milk, or sausages, and all foods which may contain ptomaines, may produce this condition, while prolonged abuse of alcohol is a frequent cause of gastritis (acute alcoholic gastritis).

Certain bacteria or bacterial poisons passed from the mouth, naso-pharynx, or throat into the stomach may also induce this condition. Gastritis may also result from many abnormal states of the system. The various acute infectious diseases are not infrequently complicated by this affection.

It should be noted, too, that certain drugs, when used for too long a period of time or in too large doses, may cause gastritis—*e.g.*, potassium iodide, sodium salicylate, quinine, etc.

But in addition to all or any of these exciting causes there usually exists a diminished resistance which may be local or general, and which permits the development of an attack.

As this disease is rarely fatal, post mortem examination is seldom available, but some cases have been studied through a gastric fistula. The chief pathological changes are the signs of acute inflammation of a mucous membrane, indicated by redness and swelling, which may be uniform or in patches. The parts nearest the outlet of the stomach are often most involved. The mucous membrane becomes covered with a glary mucus, and, here and there, there may be superficial erosions and occasionally small hæmorrhagic areas. As a result of this inflammation there is desquamation of the epithelial cells, and the parietal cells become granular or cloudy. An infiltration of leucocytes into the peri-glandular tissue is more or less constant. Leucocytes are also found with the mucus.

Signs and Symptoms.—There may be few or no physical signs. Some epigastric fulness and tenderness on pressure may be present.

In many instances an acute gastritis may be part of a general gastro-enteritis, and there may not only be gastric but intestinal symptoms. Tympanitis is then often considerable.

As a rule the onset is sudden, and symptoms of all degrees of intensity may be present, from slight nausea and discomfort to acute pain with violent retching and vomiting. *Anorexia* is a prominent symptom, and flatulence and distension are commonly present. Usually the disease is unassociated with fever. Thirst is often very urgent.

In the more acute types, as in that associated with ptomaine poisoning, fever may be pronounced, and rise as high as 104° F. The pulse is correspondingly rapid and great, even alarming collapse may accompany the condition. Other signs of auto-intoxication may be marked, such as headache, acetonæmia, and sweating.

I frequently find that, in young children, *acetonœmia* plays an important factor in the clinical picture. It is true that in almost every febrile affection in childhood metabolism is perverted and acetonæmia may be present. The characteristic odour, readily detected in the breath, and the presence of acetone in the urine, make this condition easy of recognition; but in acute infantile

gastritis, or gastro-enteritis, acetonæmia is still a more pronounced symptom. There is somnolence, extreme lethargy, and not infrequently symptoms of disordered cerebration, which are apt to give rise to unnecessary apprehension of a basal meningitis.

Many children acquire a special predisposition to repeated attacks of gastritis, often from very inadequate causes, some nervous excitement or a little laxity in the supervision of the diet or any change from the routine being sufficient to precipitate an attack.

Vomiting is more or less persistent, and usually copious. The stomach contents show little signs of digestion, even when the food has lain in the viscus for some hours.

Mucus in quantity, with some bile, and it may be traces of blood, will also be present.

Complaint may be made of the taste of the vomited material. It may be both bitter and sour, and sometimes it has an offensive odour from the presence of organic acids—especially from butyric acid. If the gastritis be due to a bout of alcoholic excess, acetic acid in considerable amount can often be recovered from the vomitus.

Most frequently there is a hypochlorhydria or free hydrochloric acid may be quite absent, due to the excessive formation of mucus and inflammatory exudation. Much less commonly a well-marked hyperchlorhydria accompanies the attack (acid gastritis).

Flatulent eructation is usually a prominent and troublesome symptom.

The bowels may be irregular, constipation or diarrhœa being present, or these two conditions may alternate.

The tongue is usually thickly furred at the outset of the disease, but later it may become red, painful, and very flabby, with its edges indented by the teeth.

Herpes labialis is quite commonly a feature of this condition.

Even when there is no general disturbance of the intestine, occasionally the duodenum becomes involved in the process, and an extension into the biliary ducts may take place and cause catarrhal jaundice.

The urine presents the features of a febrile condition. It is scanty, high-coloured, and loaded with urates. Acetone and an excess of indican are commonly present.

The attack rarely lasts longer than from three to four days.

Differential Diagnosis.—It is obvious that such a clinical picture is, from time to time, likely to give rise to a difficulty in diagnosis.

When head symptoms are severe and pronounced, intra-cranial mischief may be suspected and give rise to needless anxiety. In meningitis the onset is slower and the duration longer; the antecedent history is of some standing. The temperature in adults is usually normal, while in children it is commonly elevated. In the absence of a Kernig's sign and optic neuritis the symptoms favour gastritis; but if doubt remains, a lumbar puncture and an examination of the cerebro-spinal fluid will clear up the diagnosis.

A careful chemical and microscopic examination of the characteristic vomit will also afford valuable information as to the nature of the disease.

The vomitus and the test meal contain undigested food material mixed with an abundant glairy mucus. The mucus may be stained greenish from bilious regurgitation, or be streaked with blood, especially if the vomiting be violent.

The reaction is usually acid to litmus, owing to the formation of organic acid. Butyric and lactic acids are most commonly found.

Not infrequently HCl is absent or diminished. Occasionally a hyperchlorhydria may be present (acid gastritis). The mucus contains leucocytes.

Peritonitis and *intestinal obstruction* (Intussusception and Torsion) are associated with more pronounced abdominal physical signs and more persistent vomiting.

Biliary Colic and *Cholecystitis* may be associated with many symptoms of acute gastritis, but a careful consideration of the history and more local physical signs associated with these several conditions will usually prevent error.

Treatment.—There can be no doubt, in the more acute cases, that the most effective treatment is to encourage nature in her efforts to empty the stomach and bowel and give the digestive apparatus a rest.

Lavage, carried out early, is therefore one of the most important therapeutic procedures.

I have found that more speedy relief is got in those cases by adopting the following plan than from any other method I have tried.

The stomach should first be washed out with from ten to fifteen ounces of warm sterilized water, passed slowly through the syphon. Care should be taken to avoid sudden distension of the stomach. This fluid should then be removed, and a second quantity introduced, containing about forty grains of sodium hyposulphite in solution. In the presence of acid, organic or inorganic, the hyposulphite is decomposed with the liberation of SO_2, which acts as an excellent disinfectant and germicide. The hyposulphite solution is left in the stomach for about one minute, and then syphoned off. The stomach is finally cleansed by a third quantity of warm water, which in its turn is removed.

If lavage cannot for any reason be undertaken, and it is often inadvisable in very nervous people and young children, vomiting should be encouraged at the outset in all acute forms of gastritis.

When the disease has resulted from the ingestion of unsound food, it is desirable to have the gastro-intestinal tract evacuated of its contents as speedily as possible. An emetic of salt or mustard in tepid water may be administered, or, if more prompt and certain effect be desired, nothing is better than a hypodermic of apomorphine.

The disagreeable taste of salt or mustard will often cause children to struggle violently if an attempt to administer these be persisted in. An equal part, ʒi—ʒii, of syrup of squill and ipecacuanha wine will be readily taken, and will act as an efficient emetic. But for adults I would recommend more than all these the administration of ʒi of sodium hyposulphite in half a pint of tepid water. It is almost always speedily vomited, and if it is not, and passes out of the stomach into the intestine, it acts as a useful saline aperient and gastric antiseptic.

Later on, urgent symptoms may be dealt with.

If vomiting be persistent, sinapisms to the epigastrium are useful. Mustard and linseed (one to three) may be applied at first, and then followed by hot linseed poultices or hot fomentations. Frequent doses of hot water are always useful, and, if retained, greatly aid elimination of toxic materials. Demulcents are usually indicated, and are well borne and of distinct therapeutic value. Barley water and other mucilaginous drinks have a sedative influence on the irritated mucous membrane.

The various medicinal sedatives may also be tried after the stomach has been thoroughly emptied by lavage or emesis. The

administration of these remedies may not be necessary unless vomiting or gastric irritation be urgent.

Bismuth, precipitated calcium carbonate, cerium oxalate, and alkalies may be found helpful.

℞ Liq. Bismuthi .... ℥iiss
Acidi Hydrocyanici diluti .... ♏ 12
Sodii Bicarbonatis .... ℥ii
Nepenthe .... ℥i
Liquoris Magnesii Carbonatis .... ad. ℥vi
Signa: ℥ii, in water, every three hours. M.

℞ Cerii Oxalatis .... gr. 72
Tincturæ Belladonnæ .... ♏72
Bismuthii Carbonatis .... gr. 120
Mucilaginis Acaciæ .... ℥ii
Aquæ Chloroformi .... ad. ℥vi
Signa: ℥ii, in water, every three hours. M.

If the bowels have not acted, a large dose of calomel, gr. 5 to 10, may be given. We have no better intestinal antiseptic, and when given in a frankly purgative dose of gr. 6, repeated in one hour, as recommended by Ewald, it has a very useful influence in shortening the attack.

Thirst is often extreme, and if the frequent doses of hot water are not being retained it is excellent treatment, when diarrhœa is absent, to give normal saline enemata. From ten to fifteen ounces may be introduced into the bowel every four hours, and if the patient be collapsed ℥ii—℥iv of brandy may be added.

Diet.—As the mucosa in nearly all cases of acute gastritis is very irritable, the wisest initial course is to give no food by the mouth.

When the acute phase is over, and vomiting has ceased, small doses of whey or peptonised milk, given hourly in ℥ii doses, may be usefully employed. Later, plain milk, diluted with an equal part of lime water, can be administered frequently in ounce doses, or gelatine in one to two per cent. solution, mixed with an equal quantity of white of egg, is an effective remedy. When symptoms have subsided, some egg may be added to the milk diet, so as better to support nutrition.

In mild cases no special treatment is required at all. Abstention from food for an entire day and the administration of a purgative dose of calomel may be sufficient.

After Treatment.—Much debility may be left after acute gastritis, and the appetite may remain so long in abeyance that the patient's nutrition is not maintained.

Change of air, especially to the sea or hills, with gentle exercise and the administration of an acid bitter tonic, shortens greatly the convalescent period. Such a tonic as the following may be prescribed :—

℞ Acidi Hydrochlorici diluti .... ℥ii
Glycerini Pepsini .... ℥ii
Tincturæ Ferri Perchloridi .... ℥i
Syrupi Limonis .... ℥i
Infusi Gentianæ Compositi ad. .... ℥vi
Signa : ℥ii, in water, thrice daily, after food.

or

℞ Acidi Hydrochlorici diluti .... ℥ii
Liquoris Strychninæ .... ♏ 36
Tinct. Gentianæ Compositi .... ℥i
Syrupi Aurantii .... ℥i
Vini Quininæ ad. .... ℥vi
Signa : ℥iv in water, thrice daily, before food.

II. Phlegmonous Gastritis.

(Acute Suppurative Gastritis : Abscess of Stomach.)

This condition is of comparatively rare occurrence. It may be a primary or a secondary affection of the stomach due to bacterial infection. Although many organisms may be responsible, the streptococcus is the one most commonly pathogenic. The disease runs a rapid and fatal course. It appears to be more common in men than in women. Post-mortem it is difficult to find an ostensible lesion serving as a portal of entrance for the organism. At times the disease may be due to pyæmic infection.

Phlegmonous gastritis may be *generalised* or *circumscribed*. In the former the mucous membrane generally is swollen and infiltrated with pus ; in the latter there are local abscess formations.

Symptoms.—Great epigastric pain, sudden rise of temperature to 103° to 105° F., shivers, loss of appetite, marked prostration, and retching are the symptoms.

In a short time the abdomen becomes rigid, especially in its upper section, and the clinical picture may resemble that of the "acute abdomen" from perforation.

The diagnosis can only be made with certainty by finding pus in quantity in the vomited material. Micro-organisms may also be found in the purulent secretion.

These cases will always be difficult to diagnose, for the clinical picture is not very different from that of empyæma of the gall bladder or of acute pancreatitis. It will, thus, always be advisable when in doubt to make an exploratory operation.

TREATMENT is not successful, and can only be palliative. Full doses of morphine or codeine may be given for the relief of pain.

III. Toxic Gastritis.

Acute gastritis from the direct introduction by accident or suicidally into the stomach of corrosive caustics or poisons is usually readily recognised.

The more powerful caustic alkalies and corrosive acids are the poisons most commonly responsible for toxic gastritis, but arsenic, antimony, lead, mercury, and silver, as well as phosphorus, carbolic acid and alcohol are not infrequently the cause of this disease.

Many of these poisons act violently on the mucous membrane and cause its erosion, with the risk of perforation. The amount of damage done to the stomach depends on the dose taken, the concentration of the irritant, and very greatly on the amount of food which may happen to be present in the stomach.

TREATMENT consists in the removal, with the minimum delay, of the stomach contents. This may be done either by the action of emetics or by lavage. It is, however, sometimes an exceedingly risky procedure to insert even a soft syphon when the stomach has been seriously eroded by strong alkalies or acids. Perforation, which is one of the chief immediate dangers, may thus be brought about. If the case be seen *at once*, early lavage is by far the best treatment.

Antidotes for the special poisons must be employed, and demulcents, such as olive oil, linseed oil, starch mucilage, and the white of eggs should be administered.

Subsequently much trouble may ensue from cicatricial contraction of the œsophagus or different parts of the stomach necessitating surgical interference.

IV. Acute Infectious Gastritis.

It may hardly be necessary to give this form of gastritis a separate name, as it closely resembles the simple acute type.

This condition results from the entrance into the stomach of micro-organisms. Under normal conditions it is obvious that germs are destroyed by the antiseptic action of the gastric juice; but when the defence is down, locally or generally, organisms may become pathogenic.

Probably most cases arise from impurity conveyed in the food or drink. Bad water, milk, meat, and vegetable material or fruit in a state of decomposition may result in this form of acute gastritis.

This ailment, when occurring in children, is commonly associated with fever and other pronounced constitutional disturbances. It has been popularly called 'gastric fever.'

The symptoms of acute infectious gastritis may form part of the clinical picture of *ptomaine poisoning*.

TREATMENT is similar to that adopted for simple acute gastritis, and the indications are gastric cleansing by means of lavage and the introduction into the stomach of bland antiseptic solutions. Of these, sodium hyposulphite, one dram to the pint, borax or boric acid in the same strength of solution, or a very weak solution of permanganate of potash are all useful remedies.

A purgative of calomel should be administered early, and vomiting and pain should be treated as required. Gastric rest till the urgent symptoms pass over is also most essential.

A form of infective gastritis, characterised chiefly by fermentation, is met with in childhood, when milk forms the principal article of diet. Gaseous distension, flatulent eructation, and vomiting are the chief symptoms. The disease is to be recognised principally by the examination of the vomitus, chemically, microscopically, and by means of the fermentation tube.

V. Chronic Gastritis.

(Chronic Catarrhal Gastritis: Chronic Asthenic Gastritis: Chronic Dyspepsia.)

The synonyms, chronic gastric catarrh and chronic dyspepsia, are not fortunate or scientific terms. They indicate only some symptom of the affection, and under these names almost all forms of chronic digestive derangement might be included.

Many classifications of the various forms of chronic gastritis have been adopted by different writers :—

(1) Those based on pathological change, and (2) on altered function.

But although there are many different pathological conditions found at different stages of the disease, there are certain features common to all.

A chronic inflammatory disease of the gastric mucosa is accompanied by increased formation of mucus, with alterations in the chemistry of the gastric juice and in the motility of the stomach, and it ends sooner or later in change of structure.

The disease may be *primary*, due to various and prolonged indiscretions in eating and drinking, or *secondary* to the acute type as well as to other gastric and constitutional diseases.

Etiology of Chronic Gastritis.—Indiscretions in diet are responsible for a large number of cases. Over-eating and drinking, bolting food improperly masticated, tend to keep up chronic irritation of the stomach and, through time, to induce structural change. Excessive use of iced drinks, the quick ingestion of too hot fluids, and the abuse of tobacco and alcohol may all provoke this disease.

Again, chronic gastritis may be a complication and accompaniment of other stomach or hepatic diseases—*e.g.*, chronic ulceration and dilatation, or portal obstruction producing changes due to chronic venous hyperæmia.

Not uncommonly chronic gastritis is the sequel of the acute affection.

The anatomical changes and the clinical symptoms which characterise chronic gastritis are found as an accompaniment of many constitutional and chronic infective diseases.

Lastly, chronic gastritis is frequently associated with anæmias, leukæmia, tuberculosis, nephritis, diabetes, and many other serious constitutional diseases.

Symptoms.—No definite date can, as a rule, be given for the onset of the first symptoms. A complaint of long standing ' indigestion is usually made. The symptoms at different times and stages of the malady are so diverse that a diagnosis is difficult, nay impossible, without the aid of test meals and a study of the gastric chemistry. This will be readily understood if we accept Ewald's classification

of the clinical types of chronic gastritis based on the observations of the gastric contents after the test breakfast.

Thus we have cases in which the free hydrochloric acid is increased and where mucus is only slightly in excess: others where mucus is very excessive and the hydrochloric acid defective or absent, and others again where the test breakfast shows a complete absence of hydrochloric acid, pepsin, and rennet. The latter form of chronic gastritis will be more fully discussed in the chapter on achylia.

Gastric discomfort, rather than pain, is the keynote in the symptoms. Epigastric fulness and a sense of oppression are usually complained of after eating. After food, wind is belched from the stomach (flatulent dyspepsia), and with it eructations of sour or bitter fluid. The stomach is often distended with gas and palpitation and dyspnœa are frequent. The pulse may be slow and small. The intestines may also participate in the gaseous distension and a considerable degree of meteorism be present. Vertigo (stomachic) is also very common. There may also be a chronic pharyngitis with so-called stomach cough. The appetite is poor and capricious, and often when some desire for food is felt it is satisfied with a very small and inadequate quantity. A bad taste in the mouth is frequently a source of much annoyance to the patient, and contributes to the distaste for food. Heartburn, or Pyrosis, is not uncommon at certain stages of the disease, doubtless due to excess of organic or inorganic (HCl) acids. The tongue is flabby and large, and shows indentations from the teeth round its margins. It is coated with a thick dirty grey moist fur. The same appearance of the tongue may occur in other digestive and general diseases, and must not therefore be regarded as characteristic of chronic gastritis. The breath is usually offensive.

Nausea and vomiting very commonly are prominent features during the course of this disease. Vomiting is usually delayed, and comes on most frequently about two hours after food. It gives temporary relief.

In the gastritis of tipplers (chronic alcoholic gastritis), retching and vomiting in the early morning on getting out of bed are very characteristic features. In these cases glary mucus is the only thing brought up, perhaps with some remnants of the supper of the previous night. Associated with this vomiting, morning looseness of the bowels is a common occurrence.

In the vomiting after meals, only part of the food is, as a rule, put up, and it usually contains an excess of mucus, and frequently the food particles are badly digested.

Irregularity of the bowels is usual, and constipation is rather commoner than diarrhœa.

When much intestinal dyspepsia accompanies the condition, the symptoms may be greatly aggravated.

In the type of gastritis associated with great hypertrophy, actual stenosis of the pylorus may exist, with all the signs of gastric retention. A tumour, small in size and usually movable, may be felt in the epigastrium in many of those cases. It is then that often considerable difficulty will arise in the differential diagnosis of gastric carcinoma with a small tumour in this situation.

It is remarkable even in long-standing cases of gastritis how well the general nutrition may be maintained; but sooner or later the complexion gets muddy, dark rings form under the eyes and anæmia of varying intensity may supervene. The patient is susceptible to cold, and suffers greatly from clammy cold hands and feet. His weight declines, and other signs of auto-intoxication are rarely absent. He may become nervous and irritable, suffer from insomnia, and frequently have pain in the frontal region, mental depression and hypochondriasis.

The urine often affords ample confirmation of the digestive and general disturbance. It is scanty, and may at times be loaded with urates, but more commonly contains phosphates in excess (phosphaturia). Indican can sometimes be detected in quantity, as well as some acetone. From the prolonged auto-intoxication chronic interstitial nephritis is not a very rare sequel.

Diagnosis.—This chiefly depends on a careful examination of the stomach contents, and cannot be made from the clinical history or symptoms alone. In most of these cases contents extracted from the fasting stomach reveal an empty viscus, or one containing only mucus in excess.

No proper treatment can be undertaken till a knowledge of the digestive function, in each individual case, has been obtained.

Although the appearance of the vomit may give us considerable information, test meals should always be employed in making a diagnosis, and these should be subjected to a chemical and microscopic examination.

The Ewald's test breakfast may be given and withdrawn in one hour. In the different types of gastritis the findings will be different :—

(1) *Simple Chronic Gastritis.*—The test breakfast extracted will have the following characters :—

(*a*) Macroscopic Appearances.

1. Quantity : The return will usually be rather above the normal.

2. The toast will show recognisable parts of various sizes, indicative of imperfect digestion.

3. There will be an excessive amount of mucus incorporated with the test breakfast. The amount varies greatly in different cases. A glass rod dipped into and lifted through it will take with it a stringy mass of mucus. This tenaciousness of the gastric content will also be demonstrated by the slower rate of filtration, and often by the clarity of the filtrate. The mucus which, during syphonage, is so copiously poured out from the glands in the mouth and gullet, floats on the surface discrete from the gastric contents, and can easily be distinguished from that of a true gastric catarrh, which is intimately mixed with the test meal.

Mucus in the gastric contents, as a contamination from the gullet or mouth, is mixed with a cellular element, squamous in character, while that derived from the stomach is associated with columnar epithelium.

(*b*) Chemical Analysis.

1. Free Hydrochloric Acid.—Usually there is a well-marked hypochlorhydria (0.05 to 0.07 per cent.). Occasionally an achlorhydria is present.

2. Total Acidity is often normal, for the defect in the HCl is made up by an increase in organic acids.

3. Combined HCl is also diminished.

4. Occult blood is absent.

5. Pepsin and rennet.—These must always be examined for when HCl is absent or very defective. It is rare to find a true achylia, although when there is a marked hydrochlorhydria there may also be a deficiency in the enzymes.

(*c*) Microscopic Examination.

1. Columnar Epithelium, mixed with mucus, may be seen in quantities.

2. Organisms, yeast, sarcinæ, etc., are only present when we have gastric retention.

3. Starch Granules.—On placing a droplet of the contents on a slide and staining with a very weak solution of iodine, the starch will be found fairly well digested, erythrodextrin being scanty and achroödextrin abundant.

Variations from the above Type of Gastritis.

(2) *Acid Gastritis* (hypersthenic gastritis: gastritis glandularis proliferans).—In this type there is a hyperchlorhydria, with extensive increase of the paryenchymatous tissue. Later on there is also an increased connective tissue formation. Probably in a great many instances these cases are rather a true hyperchlorhydria than a true gastritis, or they may be regarded as only a stage of the latter disease.

(3) *Hypertrophic and Sclerosing Gastritis.*—This type of gastritis may be regarded as an advanced stage of simple gastritis.

The stomach may become very small in size, which prevents a full test breakfast from being taken. Hypertrophic and sclerotic areas may be found side by side, giving rise to a varying amount of irregularity in the surface of the gastric mucosa. There may be interference with the pylorus amounting almost to an actual stenosis with all the symptoms and signs of gastric stasis. The extracted meal will be found to be badly digested, and mixed with a varying amount of mucus, and it may contain remnants of a previous day's meal. The total acidity may be high, owing to the formation of organic acids. Organisms of fermentation and putrefaction may be found.

As this disease causes destruction of the secreting cells and interferes with the blood supply to the secreting glandular tissue, there is a progressive diminution and ultimately a complete suppression of the secretory function.

(4) *Atrophic Gastritis.*—From the outset atrophic changes may be the predominant feature.

In atrophic gastritis we have a complete arrest of all secretory function—proferments, acid, and even mucus may be absent. The test breakfast is returned, little changed, and has only the appearance of maceration. This is one of the affections in which a permanent achylia is found.

Bassler thinks that the presence of free gastric cells in the fasting stomach is characteristic of this disease.

These conditions can alone be diagnosed by gastric analysis. The characters of the test meals and the presence or absence of HCl and the ferments will determine the treatment to be adopted. The symptoms are those described under chronic simple gastritis, with variations in keeping with the changes in the gastric functions.

Pathology of Chronic Gastritis.—The changes which occur are complex, and not confined to any one tissue. At first the glandular tissue and mucous membrane are principally involved. If the disease be protracted, changes in the interstitial tissue follow later on.

Two types of chronic gastritis are commonly described :—

(1) *Simple Chronic Gastritis.*—This condition is associated with pallor of the mucous membrane, to which is adherent tough mucus. The stomach is enlarged. At the pyloric end the mucous membrane may become rough and pigmented, and warty looking, giving rise to the *état mammeloné* of the French. The mucous membrane is sometimes thickened, but may become thinner and less friable than normal. There are both parenchymatous and interstitial changes. There is an abundant leucocytic infiltration near the surface of the mucosa, and from the sub-mucus layer extensive connective tissue formation passes between the glands.

(2) *Sclerotic or Fibrotic Gastritis.*—This condition is nothing more than an advanced stage of the simple type.

There may be complete atrophy of the mucous membrane, with entire loss of its normal function. There is enormous thickening, sometimes observed and due to (1) increase in the muscularis mucosæ, or (2) to new connective tissue formation, giving rise to the appearance of a diffuse carcinoma (Hypertrophic Gastritis). In this type the stomach may be very small, due to contraction of the connective tissue (cirrhosis).

In some cases hyperplasia of the parenchymatous tissue occurs either diffusely or confined to certain areas, particularly towards the outlet. Such cases are, in the early stage, those that produce an excessive secretion of hydrochloric acid with symptoms of hyperchlorhydria, and constitute the *acid gastritis* of Boas.

When connective tissue formation begins, atrophic changes in the specific glandular secretory tissue follow in time. Ultimately the mucous glands predominate, and, when the stomach is opened, give rise, by their excessive secretion, to the appearance of an abundant tenacious coating adhering to the mucous membrane.

Atrophic Gastritis is a later and terminal stage of chronic gastritis ; or it may start from the outset insidiously as the atrophic type, resembling in this respect a primary sclerosis of the kidney.

Prognosis in Chronic Gastritis.—If the disease be treated early, before organic change in the mucosa has begun, the prognosis is not unfavourable ; but when structural change has advanced and the hypertrophic or atrophic type of gastritis has become established, palliative treatment is all that is possible.

Treatment of Chronic Gastritis.—A chronic affection of this kind, associated frequently with organic change in the stomach, and often with a disturbance in its chemical and motor functions, cannot be otherwise than troublesome to treat. Indeed, it is this class of case which offers the enormous clientèle to the quack, who promises speedy relief from the symptoms which have, by their persistence, exhausted the patience of the sufferer.

No cases require more careful study and no means of arriving at a diagnosis as to the type and stage of the disease should be left unexplored. It is only thus we may expect, with any degree of confidence, treatment to be palliative or curative. It is commonly the case that patients who are suffering from chronic gastritis are, because treatment fails and symptoms persist, often described as neurotics and neurasthenics.

Nor must we rest content with any one method of treatment, for in the more severe cases we may require to employ numerous therapeutic agencies to effect improvement.

The more important therapeutic measures for the treatment of chronic gastritis comprehend all our modern methods. Dietetic, medicinal, climatic, electrical, and psycho-therapeutic procedures may all require to be invoked ; while many of the manipulative methods, including lavage, massage, hydrotherapy, physical culture, etc., may all be used with advantage.

One of the most important considerations to keep in mind is that every case must be treated on its merits and in accordance with the data yielded by a full examination of the patient.

In chronic gastritis routine treatment is fatal to success.

Prophylactic Measures.—As chronic gastritis may be the sequel to repeated attacks of acute gastritis, or acute dyspepsia, a great effort should be made by the practitioner to prevent the recurrence of these. Most frequently acute dyspepsias arise from

an ill-regulated mode of life, lacking in wholesome exercise and good hygienic conditions.

A healthy mouth, with sound teeth, should always be secured; all septic conditions of the upper passages should be eliminated.

Thorough mastication and insalivation should be insisted upon. To aid this it may be well to advise, for all people who have acquired the habit of eating too quickly, a dry diet with fluids to be taken after all the solids have been disposed of.

All obvious abnormal conditions should be, as far as possible, removed. If constipation be present it must be corrected, and all excess in diet, alcoholic beverages and tobacco ought to be interdicted.

Open air exercise should be encouraged, and some form of home physical culture should be insisted on. Provided the patient has a good reaction, cold baths, or cold sponging, every morning are useful.

If nervous symptoms are prominent, benefit may be obtained from high frequency current.

Treatment of the Confirmed Condition.—As nearly as possible these will be considered in the order of their importance.

Having adopted all the prophylactic measures that the case dictates, there remain the following methods of treatment:—

1. *Dietetic.*—Here no hard and fast procedure is possible. According to the observations we have made of the chemical and motor functions of the stomach must we be guided.

In the aberrant cases of gastritis acida, with hyperchlorhydria, probably the main indication is the control of the acidity for a period of a few weeks by an egg-switch diet and suitable alkalies.

For those cases in which we have hypertrophic changes with stasis of the gastric contents, lavage and small fluid meals will be most suitable.

In the atrophic types, with total achylia, the treatment will be discussed under *achylia gastrica.*

But for all of the ordinary cases, without gross anatomical change, constituting the vast majority, the following outline of treatment may be adopted:—

If vomiting be a prominent feature and much pain and discomfort be present, the meals should be small and frequent, and entirely liquid. Milk and lime-water, buttermilk, barley water, chicken

soup, egg-switch, and Benger's food are among the articles of diet which may be tried.

About four or five fluid ounces may be given every two hours during the day. The mouth should be rinsed with an antiseptic lotion before each meal, and the nutriment should be sipped very slowly.

Later on, when irritation is diminished, the meals may be increased in quantity and given less frequently, and some semi-solid or solid material added—*e.g.*, custard pudding, curds and cream, sago soufflé, scrambled egg, chicken and fish creams.

Even when the symptoms are temporarily allayed we must recognise that we are dealing with a chronic disease which will require prolonged, if not permanent, treatment.

A dietary of a digestible character must be prescribed, and a consideration of the essential points referred to in the chapter on dietetics will enable the physician to outline for his patient a suitable scheme of alimentation.

As a rule a dry dietary, consisting of three meals per day, will be found most suitable for patients suffering from chronic gastritis, and whose symptoms are not urgent. It is well, too, for such cases to have at least five hours' interval between the three repasts, and a volume of some suitable fluid, sipped slowly, two hours before each meal. The fluid may either be cold or hot water, some of the mineral waters, as Vichy, bouillon or weak tea.

The three principal meals should be very simple, and never composed of many courses.

Bread should always be stale or toasted, and rusks and unsweetened biscuits are almost always suitable. The lighter and more delicate vegetables such as spinach, asparagus, vegetable-marrow and cauliflower, when well cooked, will form a useful addition, in small quantities, to the dietary. As a rule strong tea, coffee, and alcohol are contra-indicated.

Various writers have framed special diets for such cases. The objection to any routine is that the diet may be distasteful to a certain individual, and it is well to remember the proverb that "what is food for one may be poison for another."

Einhorn recommends the following :—

8-0 a.m. Two eggs, 60 grm. of white bread, 15 grm. butter, one cup of tea with 10 grm. of sugar.

10-30 a.m. 250 grm. koumiss, matzoon, or milk, 30 grm. crackers, with 20 grm. butter.

12-30 p.m. 2 ozs. fillet steak or white of chicken, 100 grm. mashed potato or boiled rice, 60 grm. white bread with 15 grm. butter, one cup cocoa.

3-30 p.m. Repeat 10-30 a.m. meal.

7-0 p.m. Farina, hominy or rice boiled in milk, one small bowlful, two scrambled eggs, 60 grm. bread with 15 grm. butter.

Ewald has a diet not dissimilar to above, as follows :—

8-0 a.m. 150 to 200 grm. tea, with 75 to 100 grm. stale white bread, toast, or rusk.

11-0 a.m. White bread 50 grm., with butter 10 grm., cold meat or ham 50 grm., one-third litre of milk.

2-0 p.m. 150 to 200 grm. water, milk, or bouillon made from a white flesh, 100 to 125 grm. meat or fish, 80 to 100 grm. vegetables, 80 grm. of mixed fruit.

4—5 p.m. one-fourth to one-third of litre of warm milk or café au lait.

7—8 p.m. 200 grm. soup or rusk sop, 50 grm. white bread, with 10 grm. butter.

10-0 p.m. If it is thought necessary and advisable, one cup of tea with 50 grm. of white bread, rusk, or biscuit may be given.

If these special diets are not adopted we can have recourse to the following :—

Boiled mutton or grilled under-done fillet steak or cutlet are the lightest forms of flesh food. White fish, steamed, or stewed in milk, are also suitable, and fresh eggs cooked lightly or beaten up with a small quantity of milk are well borne by most patients. Thoroughly boiled macaroni may be eaten freely, and a dry potato, if well masticated, is permissible.

2. *Lavage.*—This procedure often yields excellent results. It should be carried out with care for a limited time, but if improvement is not obvious it ought to be abandoned. For full details of the method see chapter on Lavage, page 98.

3. *Regimen.*—A life spent in the open air, with a moderate amount of exercise, will greatly aid treatment. When this is impracticable

it is often difficult to retain the digestive functions in a normal state in all those patients who lead a sedentary city life. For people whose occupations preclude out-of-door exercise a great deal can be done to promote general and digestive health by some systematic home physical culture. For this purpose the exercises which I prefer most are those of Müller.

Change of climate and residence in some of the health resorts which offer special treatment for digestive derangements can often with advantage be recommended. Many of the Spas, where natural alkaline mineral waters are found, offer the advantages of change of climate, environment, and diet combined, with the therapeutic influence of the mineral water. For the most part, in those cases of chronic gastritis where mucous catarrh is a prominent feature, the alkaline and saline springs are indicated. These have the power of thinning and dissolving the tenacious mucus present in the stomach, and they may stimulate the secretion of gastric juice (HCl) which is defective or absent. Thus for the ' cure ' by a saline water patients may be sent to Carlsbad, Kissingen, Homburg, Weisbaden and Vichy. If the patient cannot leave home he may employ many of these waters in his own residence, as quite a number of them are bottled at the Spas and imported. Except for uncertain climatic conditions, all the advantages of the foreign spa may be had in our own country at Bath, Harrogate, or Strathpeffer.

The prolonged use of a compress, wrung as dry as possible out of hot water, and applied to the gastric region, I have found of very great service. For patients not confined to bed, and who may be following their avocations while under treatment, it can be worn during the entire night.

4. *Medicinal.*—As an adjunct to the above measures medicinal treatment is advantageous.

The exact medicine to prescribe will be indicated mostly by the findings of the test meal.

If hydrochloric acid be defective in amount, it may be supplemented, according to the degree of deficiency, by the administration of varying doses of dilute hydrochloric acid of the B.P. It is doubtful if the addition of pepsin is of any great advantage, for it is frequently present in adequate amount, even though the HCl be deficient. Its addition, however, can do no harm.

The appetite is improved by the use of bitters. Of these the more useful are Nux Vomica, Gentian, Calumba, and Condurango. For

example, some of these formulæ may prove useful when acid is defective :—

℞ Acidi Hydrochlorici diluti .... ℥iv.
Tincturæ Condurango .... ℥vi.
Liquoris Strychninæ .... ℥iss.
Infusi Calumbæ .... ad. ℨvi.
Sig. :—℥ii, in water, t.i.d. p.c. M.

℞ Acidi Hydrochlorici diluti .... ℥iv.
Tincturæ Gentianæ Comp. .... ℥vi.
Tinct. Capsici .... ℥ss.
Syr. Aurantii .... ℨi.
Infusi Quassiæ .... ad. ℨvi.
Sig. :—℥ii, in water, t.i.d. p.c. M.

℞ Acidi Hydrochlorici diluti .... ℥vi.
Tincturæ Cinchonæ Comp. .... ℥vi.
Tincturæ Condurango .... ℥vi.
Tincturæ Zingiberis .... ℥ii.
Syr. Limonis .... ℨiss.
Aquæ Menthæ pip. .... ad. ℨvi.
Sig. :—℥ii, in water, t.i.d. p.c. M.

Instead of using HCl after meals as a direct supplement to the gastric juice, we may sometimes obtain the same result, if the peptic glands are still intact and their function only in abeyance, by the administration before each meal of—

Sodium Bicarb. .... gr. 5
Normal Saline .... ozs. 2

This solution is a useful stimulant of the gastric glands, and may promote an increase in the output of HCl.

If analysis shows that acid gastritis is present, alkaline sedatives are indicated. Should there be eructations of gas, and any tendency to nausea or vomiting, sedative carminative remedies are useful. Of these, Bismuth, Magnesia, Cerium, and Sodium Bicarbonate, combined with some antiseptic remedy, will often give relief to the patient.

The physician should not encourage the constant use of sodium bicarbonate, although it may give speedy temporary relief in cases of super-acidity. It is harmful in large doses because it may not

only neutralise but alkalise the gastric contents and so derange the normal intestinal digestion. Small doses of sodium bicarbonate have, on the other hand, an after-stimulant influence on the acid secretion. Bismuth and magnesium carbonates are free from these objections. The prescriptions given for peptic ulcer are also suitable for the treatment of acid gastritis, or this formula may be used :—

℞ Liquoris Bismuthi .... ℥ii.
Tincturæ Belladonnæ .... ℥ii.
Resorcin .... ℥ss.
Liquoris Magnesii Carbonatis .... ad. ℥vi.
M.

Sig. :—℥ii, in water, thrice daily, after food.

There may be many special symptoms in individual cases which may require special treatment. At all times it will be of great importance to overcome any coincident constipation. On account of the gastric derangement we are usually precluded from giving a rough dietary which might have a laxative effect. Thus we may have to rely on some gentle laxatives—*e.g.* :—

℞ Extracti Sennæ Fructūs Liquidi.... ....
Cascara Evacuant .... āā. ℥vi.
Tincturæ Podophylli .... ℥iss.
Decocti Aloes Compositi .... ad. ℥vi.
M.

Sig. :—℥ii, in water, morning and evening.

℞ Extracti Euonymi Sicci
Extracti Cascaræ .... āā. gr. ½
Extracti Belladonnæ
Aloini .... āā gr. ¼
M.

Ft. Pil, Mitte tales 24.

Sig. :—One pill, twice or thrice daily, as may be required.

Electrical treatment, by high frequency current, has its advocates. I have not had nearly so much success from its employment as from thorough general massage.

CHAPTER XVIII.

PEPTIC ULCER (ULCUS VENTRICULI: ROUND ULCER).

Etiology and Pathology.

Gastric, Pyloric, and Duodenal Ulceration.—In many instances it is impossible actually to locate the precise situation of the ulcer. It is not at all surprising that sometimes a duodenal ulcer cannot be differentiated from a pyloric ulcer, or an ulcer inside the viscus, but near to the pylorus, from a pyloric ulcer. Symptoms and signs are at times aberrent. Moreover ulcer is often present when it is not diagnosed clinically, and a great many so called cases of "recurrent dyspepsia" are due to this cause.

Etiology.—Our former views as to the occurrence of peptic ulceration have been largely modified by more scientific and accurate investigation and by the result of surgical procedures.

Formerly it was considered that peptic ulcer was largely confined to young and chlorotic women, but we now recognise that while ulcer *inside* the viscus may still have a rather higher incidence in women, duodenal ulcers are far more common in men.

Unhealthy occupations and bad hygienic surroundings promote digestive derangements which make for ulceration.

Predisposition plays a large part in gastric and duodenal ulceration. The disease has a distinct tendency to be familial and to attack several members of the same family.

As far as we know, the etiological factors promoting peptic ulcer are the same, no matter where the lesion be situated.

It may at once be said that *a peptic ulcer never forms outwith the range of action of the gastric juice.* It would seem, therefore, obvious that the gastric juice plays an important rôle in the production of these lesions, and that at least without its intervention no true peptic ulcer can form. The name *peptic ulcer* expresses the fact that these erosions are due to the peptic action and autodigestion of the gastric mucosa.

It will, however, be at once seen that there are difficulties in accepting this view as a complete explanation of the pathogenesis of a gastric or duodenal ulcer.

Clearly other factors must co-operate with the action of the gastric juice, and some light may be thrown on this subject by a consideration of the causation of so-called *post mortem* ulceration. When the circulation of the alkaline blood through the gastric capillaries ceases at death, the presence of gastric juice in the stomach suffices, and very rapidly, to produce widespread digestion of the mucous membrane. Some protective influence against the destructive action of the digestive fluid has been withdrawn at death, and inferentially we must look for the advent of certain factors which, during life, may permit of a similar pathological process to occur.

In health, in all probability, many factors are at work to prevent ulceration.

Hyperchlorhydria is undoubtedly one of the conditions which favour the production of ulcer. It is true that in the great majority of cases hyperchlorhydria is present, but from time to time one finds, by exception, that peptic ulcer coincides with normal or even hypo-acidity. Statistics on this point vary so greatly as to be of little value in elucidating the subject.

The gastric juice of a dog possesses a very high acidity, and yet this animal does not seem to be specially liable to peptic ulcer.

An extremely high percentage of HCl in the gastric juice, especially when secreted into the empty stomach, must have an irritating effect amounting to a mild form of trauma.

But even though this be admitted, it is not certain whether the auto-digestion of the stomach is caused by the HCl or the pepsin. In every case where there is an aberration in the amount of free HCl there is not necessarily a corresponding alteration in the amount of pepsin present.

In the same individual the amount of free HCl varies greatly under different conditions. To effect digestion not only must there be free HCl but pepsinogen (=pepsin). It might reasonably be claimed that ulceration was as much due to the pepsin as the acid.

Still it is a safe rule to keep in mind that a well-marked hyperchlorhydria is usually a pre-clinical stage or accompaniment of ulceration. This rule holds good so often that it is impossible not

to accept the view that there is a close relationship between the disorder of secretion and the process of ulceration.

It is true also that the gastric mucosa in health acquires, from long habituation, a resistance to the action of HCl, and that one of the recognised complicating sequels to a gastro-enterostomy may be the formation of a peptic ulcer at the stoma in the jejunum, from the absence of acquired immunity to unneutralised gastric juice.

After a gastrostomy, unless the valve-like aperture prevents much escape of gastric juice, there is also a considerable degree of inflammation of the skin around the external wound, which may end in the formation of ulcers. These show a tendency to extend and to form so many " round ulcers."

Many people, however, suffer from super-acidity for years, and yet never develop ulcer. On the other hand, it is held by Ewald, and other observers, that the hyperchlorhydria and over-secretion result from the presence of ulcer, and that these do not necessarily ante-date the lesion.

While then it is certain that peptic ulcer never forms outside the range of action of gastric juice, we may certainly say that auto-digestion alone is not the real pathogenic factor. Indeed, it may be claimed that hyperchlorhydria, by enhancing the antiseptic action of the gastric juice, inhibits the activity of septic organisms, and should diminish rather than increase the tendency to ulceration.

Perhaps more important is the influence of gastric stasis in the formation of ulcer. If highly acid and fermenting contents are retained too long in the stomach, then irritant and toxic action may induce ulceration. In gastric, as distinguished from duodenal, ulceration, the most recent work tends to show that the stomach empties more slowly than is normal. This is due to some hypertonus and remnants of a meal found in the stomach six hours after ingestion is in favour of ulcer.

The living healthy mucous membrane undoubtedly resists ulceration, but the injured mucosa may succumb. And yet experimental trauma is rarely followed by ulceration, but rather by rapid healing. Indeed, the operation of gastro-enterostomy could not be performed with safety if the injured mucosa were to be digested by the action of the gastric secretion. These considerations seem to indicate that the influence of trauma is uncertain as a pathogenic factor in the production of peptic ulcer.

Trauma need not be gross or direct to play a rôle in predisposing to ulcer. Tight-lacing and occupations involving postures in which the abdominal organs are subjected to pressure may also predispose to peptic ulcer.

Circulatory disturbances may, in certain cases, determine ulceration. Auto-digestion of the stomach occurs, as we have seen, rapidly after death and the cessation of the circulation. Necrosis quickly follows thrombosis or the obstruction produced by emboli in the gastric vessels. Experimental erosions can be brought about by the introduction of wax emboli. There seems, however, to be a marked difference between the experimental and spontaneous ulcer. The former heals quickly, the latter slowly.

Peptic ulcer has been seen to result from disease in the vessel walls. Arterio-sclerosis or endarteritis with thrombosis causes a local loss of blood supply which ends in ulcer by the digestive action of the gastric juice. But ulcer is uncommon in the elderly when vascular disease has its highest incidence. It is thus not possible to claim local vascular disease as a prominent pathogenic factor in the formation of peptic ulcer.

It may well be, too, that in the most contracted part of the stomach, where the mucous membrane is thick and thrown into deep folds, and when inflammatory mischief is at work, as in acute gastritis, there may be a sufficient amount of pressure exerted on certain parts from tumefaction to produce ischæmic anæmia and necrosis, proceeding, later on, to ulceration.

There is a general consensus of clinical experience which leaves little doubt that anæmia and chlorosis are apt to be associated with gastric ulceration. Anæmia, experimentally produced, has been followed by this lesion. The relationship between anæmia and peptic ulcer is not, however, quite simple. In the majority of cases of chlorosis there is an accompanying hyperchlorhydria, and it is not improbable that the vitality and resistence of the gastric tissue, in common with other tissues, is rendered less adequate by the bloodlessness. Auto-digestion may thus take place.

It may also be that certain aberrations in the innervation of the stomach may induce the process of ulceration. These include injuries and diseases of the spinal cord or lesions of the splanchnics, solar plexus, or vagi. In all probability the ulcer which sometimes follows severe burning of large tracts of the skin comes under this category.

Another potent influence in the predisposition to ulcer is the toxic factor. Toxic agents, whether organised or unorganised, may determine ulceration. In the severe form of auto-intoxication of uræmia it is not an uncommon feature to find that the vomit is blood-stained, and that *post mortem* the gastric mucosa shows minute superficial erosions. It is, however, true that in such cases other factors may be responsible for the ulceration.

Microbic infection of the gastric mucous membrane, from a great variety of causes, is probably responsible for many ulcers. Against the view that organisms are capable of inducing ulceration is often advanced the high antiseptic power of the gastric juice, especially when it contains an excess of HCl. But it should not be forgotten that in healthy conditions of the stomach, during the period of fasting, little or no gastric juice is present, and that it is possible to have infection of the mucous membrane during these quiescent periods.

Much attention has recently been drawn to the influence of streptococci on the gastric mucous membrane, and undoubtedly these organisms are frequently the cause of ulceration. Pyorrhœa alveolaris is a fruitful source of infection, and chronic discharges from the throat and nasal passages may also have a pathogenic influence. According to the recent observations of Hurst, oral sepsis and appendicitis are the most potent pathogenic factors in producing ulcer.

In the smaller curvature, where ulcer inside the viscus most commonly occurs, we have abundant lymphoid tissue which is susceptible to infection, necrosis and ulceration. The septic origin of gastric ulceration is, in many cases, certain even when no ascertainable portal of infection can be demonstrated.

If we look at this subject from another aspect, illumination may be obtained by considering how ulceration is prevented from occurring in health ? What, if any, are the protective influences at work ? If these exist, are they local or general ?

The principal local protection is afforded by the vital protoplasm of the cells of the mucous membrane itself, and by the mucus which it secretes. When the stomach is empty the fundal end and larger curvature contract to form a tube-like structure with the formation of many rugæ. The mucous membrane thus thrown into folds appears to be in a position to offer more resistance to the ulcerative process. At the fundal end, from the more abundant mucous

membrane, there is a corresponding more abundant secretion of mucus which coats the mucosa and acts as a very powerful protection against the corrosive influence of gastric juice and the toxic influence of organisms.

The habitual upright posture during the period of gastric activity also relieves this portion of the mucosa, and doubtless has a relationship to the comparative immunity of the fundus to ulceration.

In gastric and duodenal ulceration, with hyperchlorhydria and over-secretion, there is usually an accompanying *amyxorrhœa* (diminution or absence of mucus) and the mucous membrane, by loss of this protective material, becomes the more vulnerable to the action of the gastric juice. At the pyloric end or smaller curvature of the stomach the mucous membrane shows no redundancy and correspondingly fewer rugæ and less mucus. Here it is that peptic ulcer has its highest incidence.

By far and away the most common situation for the peptic ulcer is on the posterior wall of the smaller curvature. In this situation we have the largest number of glands secreting hydrochloric acid and the greatest amount of acidity, and it is here that the chyme and gastric juice are in closest contact with the mucous membrane, both during the erect and recumbent postures. Here also abounds the susceptible lymphoid tissue.

In addition to these factors, Hurst believes that a certain type of stomach determines the exact site of the ulcer. The hypertonic, the type prevailing in duodenal ulcer, stomach predisposes to duodenal ulcer, and the hypotonic to gastric ulcer.

These anatomical and physiological conditions determine the great frequency of peptic ulcer at the smaller curvature and at the outlet of the stomach.

It has also been suggested that there might be an antipepsin which inhibits the digestive action of the gastric juice on the mucous membrane. Whether this body really exists, and, if so, whether it is manufactured locally or circulates in the blood, has not yet been accurately determined. Weinland believes that he has isolated an antiferment which he asserts has an inhibiting influence on the action of pepsin. An antiferment has been found also by Blum and Fuld in the gastric juice.

Probably the most potent protective agent against the action of gastric juice is an active and healthy circulation of the neutralising alkaline blood.

Pathological Anatomy.—Ulcers may assume many forms. For the most part they are round or oval. While commonly single, they are not infrequently multiple.

Generally speaking, ulcers may pathologically be classified as acute and chronic, and their appearances differ accordingly.

The *acute ulcer* is formed quickly, and the necrotic process is rapid, not permitting time for a large cell infiltration. They are usually soft, with well-defined and slightly rounded edges which may be firmer than the surrounding tissue. The floor of the ulcer is smooth and covered with stained mucus or altered blood, and it tends to pass deeply into the tissue and assume a punched-out appearance. It may expose the muscular or serous layer, and even its base may be formed by some organ to which it has become adherent. Acute ulcers are commonly small in size.

The symptoms of these acute ulcers need no lengthy description. They may be latent, and declare themselves suddenly by a violent hæmorrhage or by perforation. Or there may be a short period of acute indigestion with pain, nausea, and flatulence.

After rest and treatment these ulcers may heal quickly, and recovery may be permanent, or relapse may take place and the ulcer pass into the chronic form.

The Chronic Ulcer.—Unlike the acute ulcer, which is usually more or less round, the chronic ulcer assumes irregular shapes. It shows little or no tendency to heal, or it may show the process of healing and extending simultaneously. In many cases it would appear to be the direct continuation of the acute or recent ulcer. Thus it is frequently hard at parts, from connective tissue formation, and has sloping edges. It is associated with a varying amount of perigastritis and adhesion to neighbouring parts. It runs a variable course, with remissions and relapses, and may ultimately become the seat of cancerous infiltration. Such ulcers are commonly found in the duodenum or at the outlet of the stomach, and principally in men.

When an ulcer is very chronic, a degree of tolerance may be established, so that the more urgent symptoms may be in abeyance and lead to a wrong diagnosis. I have notes of a number of cases where such ulcers were associated with symptoms so slight as to be almost latent, and where the true nature of the condition was diagnosed from the laboratory findings and verified later at the operation.

When a round ulcer heals, characteristic scars are formed on the walls of the stomach. These are radiating or stellate in disposition. In the case of the more chronic indurated ulcer the result of healing is a more irregular and variable scar associated with all degrees of deformity and contraction of the stomach walls. It is thus we get pyloric stenosis, hour-glass contraction and, later, other gastric deformities.

When the chronic ulcer penetrates deeply into the gastric wall, involvement of the serous surface takes place. The resulting perigastritis is frequently the cause of troublesome adhesions which may perpetuate symptoms long after the ulcer has healed.

If healing does not take place, hæmorrhage or perforation may occur from erosion of one of the vessels or from necrosis of the entire thickness of the stomach wall. It should be noted that ulcers on the anterior wall are far more liable to perforate than those situated posteriorly. This is due to the absence of organs to which the peritoneal surface of the stomach may become adherent, and to its greater freedom of movement anteriorly than posteriorly.

Classical Symptoms.

Although it is true that most gastric ulcers are associated with more or less classical symptoms and a fairly definite clinical picture, it should never be forgotten that symptoms may be so slight as to be entirely overlooked. Failure to recognise the true condition is largely due to a too great dependence on the so-called classical symptoms and a neglect to seek for evidence of ulceration by a proper examination of the patient and a careful investigation of the gastro-intestinal chemistry.

In recurrent cases of dyspepsia or indigestion it is well to exclude ulceration before concluding that the case is functional or neurotic. In most cases of established ulceration there is a more or less prolonged history of dyspeptic disturbance.

Sometimes it is not easy, nay, occasionally impossible, to differentiate duodenal from gastric ulcer. But a failure to recognise the situation of the ulcer is of less importance than the recognition of its presence.

The symptoms and signs of gastric and duodenal ulceration are, in the main, very similar. The differential diagnosis is dependent on certain special features which, when typical, are easy of recognition.

No separate description of the symptoms of acute and chronic peptic ulcer need be given. It will, however, be noted that in the former the onset may be sudden and ushered in without previous warning by an alarming hæmorrhage or by perforation.

Pain.—Perhaps this symptom is the most constant and diagnostic in peptic ulcer. It has varying degrees of intensity and, from time to time, by exception, may be absent or so slight as not to elicit remark.

The Cause of Pain in Peptic Ulcer.—It is commonly assumed that the pain is principally due to the irritant action of solid food particles on the broken surface and exposed nerves, because it is usually worse after a rough solid meal than after a fluid diet. But pain frequently is severe when the stomach is quite empty, and food often relieves it at least temporarily. It should be remembered also that the hollow viscera are rather insensitive, at least in their mucous membranes. The second stage of colotomy, to quote a familiar example, is performed painlessly when the opening for the establishment of the artificial anus is made. Does the pain of gastric erosion owe its origin to the contact of food with the ulcerated surface? It may be said at once that this is not its principal cause. The entrance of solid, and to a less extent of certain liquid, foods into the stomach is followed immediately by a proportionate amount of distension and by secretion and peristalsis. These go hand in hand.

The pain may be regarded, in part at least, as a neuritis due to irritation of exposed nerve filaments from the surrounding inflammation or infection of the ulcer.

When vomiting takes place the pain of ulcer usually ceases quickly, because peristalsis and secretion cease. Active peristalsis involving movements of the ulcerated area is the principal cause of pain.

Persistent and severe localised pain in gastric ulcer has considerable significance. The most sensitive part of the stomach wall is its peritoneal covering. In all ulcers of some standing, which penetrate deeply and tend to perforate, a perigastric inflammation, or local peritonitis, arises. The involvement of the inflamed or adherent peritoneum in the peristaltic movements is chiefly responsible for the pain. Severe and persistent local pain may, therefore, be regarded as a danger signal of impending perforation from deep ulceration.

The relationship of pain to the degree of acidity is also of interest. Normally a high acidity coincides with active motility. In peptic ulcer hyperchlorhydria is the rule. In investigating cases of gastric ulcer a history of rapid relief from pain, after a dose of alkali, is quite commonly given. Most patients soon discover this for themselves, and seek relief from a teaspoonful of baking soda dissolved in a tumblerful of hot water. When the gastric content is neutralised by soda, although the chyme is still in the stomach, the pain ceases. It is therefore apparent that the pain is not caused by the mechanical action of the food but by the acidity which provokes and maintains peristalsis.

Over-acidity and the irritation of the ulcer promote hypertonicity of the gastric muscle, passing on to contraction and sometimes to actual gastric spasm. All agents which allay muscular contraction tend also to subdue pain. It is thus that warm applications to the gastric region often bring quick relief.

The great variation in the degree and continuity of pain complained of, in different individuals, is related to the temperament and the condition of the nervous system.

The Character of Pain.—The pain is usually described by such terms as gnawing, burning, or boring. It may be, from the time of its onset after food, persistent and fixed till relief is got by the emptying of the stomach by vomiting or through lapse of time. Or the pain may be paroxysmal, and it may be aggravated by the pressure of corsets or tight clothing.

The Amount of Pain, as a rule, is closely related to the amount and quality of the diet. Liquids generally cause less distress than rough solids, as mechanical stimulation of the stomach is greater after a mixed solid repast than after a fluid meal. Pungent ingredients, salt, condiments, and alcohol often evoke pain violently and rapidly.

The Locality of the Pain of peptic ulcer may vary with the position of the lesion. A careful location of the site and distribution of the pain is therefore sometimes, though not always, of value in localising the ulcer. It is most commonly complained of in the mid-epigastric region, a little below the ensiform process, because the most common situation of the ulcer is in the smaller curvature of the stomach. But pain may be localised to the left or right of the middle line when the ulcer is in the fundal or pyloric and duodenal ends respectively.

The pain may be confined to the gastric region in front, or may be referred to varying positions in the back. Quite commonly the pain in the back has a close relationship, in its localisation, to that in the gastric region. It may be in the middle line, or somewhat to the right or left in the dorsal region. Not infrequently it is referred to the shoulder blades. But posteriorly, its most typical situation is about one inch to the left of the spine in the small area occupied by the eleventh and twelfth ribs.

Very deep-seated persistent pain in the back is, I have found, not uncommonly associated with ulceration which is involving the pancreatic tissue.

Corresponding to the areas of pain and tenderness there may be hyperæsthetic localities of the skin. These over-sensitive areas can be located by lightly stroking the skin with the finger or pencil, when pain and wincing may be evoked by the lightest pressure.

But the pain of peptic ulcer may be complained of in less common situations. I have seen cases diagnosed as true or false angina pectoris which really were cases of gastric ulcer. Anginoid pains are not infrequently due to this cause. In the absence of cardio-vascular changes, in young subjects especially, precordial pain should not be too hurriedly diagnosed as angina or anginoid till duodenal or gastric ulcer has been considered.

Precordial pain occurs also at times in pyloric stenosis. Spasm of the pyloris is apt to be associated with anginoid pain and collapse. Some years ago I saw such a case in consultation where a boy of nine years of age had been for long confined to bed and forbidden to play any games on account of recurrent attacks of severe precordial pain supposed to be angina pectoris. There were no cardio-vascular symptoms, but pronounced signs of malnutrition were evident. An investigation revealed a dilated stomach with a marked degree of pyloric stenosis. Operation was advised and, after a gastro-enterostomy, complete and permanent relief was obtained and a rapid and excellent improvement in the nutrition and general development took place.

The time of onset of the pain in peptic ulcer is rather instructive. In ulcers well inside the viscus, the pain may commence very quickly after ingestion. It may only gradually increase in severity, or it may be acute from its commencement. In pyloric and duodenal ulcer the pain is usually delayed from two to four hours after food has been taken.

Certain writers have described the pain which habitually comes on two to four hours after food as pathognomonic of duodenal ulcer, and have called it ' hunger pain.' This so-called hunger pain is, however, only a feature of excessive secretion (gastro-succorrhœa and hyperchlorhydria), and in my experience is by no means diagnostic of duodenal ulcer. It is true that supersecretion is more commonly found in connexion with duodenal ulcer than with any other gastric affection; but it is also found as a functional derangement. When secretion of a highly acid gastric juice takes place in an empty stomach it acts as an irritant to the mucosa and ' hunger pain ' results. As soon as food is introduced into the stomach the acidity is ' combined ' and the pain quickly subsides.

It is thus, too, that in duodenal ulcer nocturnal pain (from 2 to 4 a.m.) is common. It may also occur in derangements of secretion, but until ulcer has been excluded it should be regarded as a suspicious symptom.

The mode of relief from pain is also informing. As has already been pointed out, vomiting is usually followed by a cessation of the pain. When vomiting does not take place spontaneously the patient often induces it by tickling the pharynx, with the finger or a brush, so as to ensure speedy relief. Most patients also quickly learn that the dilution of the gastric contents with hot water, especially if strongly alkalised by sodium bicarbonate, will also relieve or subdue the pain.

It is also recognised, in those cases where the pain is delayed (duodenal and pyloric ulcer), that it may be temporarily relieved by taking more food. This is very suggestive of duodenal ulcer. The cessation of the pain is due to the closure of the pyloric orifice, to the combining of free hydrochloric acid with the food, and to the lessened peristalsis which results. When the acidity later on increases, the pain gradually returns and is worse at the height of the acid tide.

The patient sometimes gives a history of obtaining relief from pain by altering his posture. When the acid contents of the stomach fall away from the seat of the lesion the pain may subside. The decubitus giving greatest relief will naturally be assumed by sufferers from peptic ulcer.

Warm applications, which cause muscular relaxation and relieve contraction and spasm, diminish or relieve pain.

Corresponding to the painful areas, tenderness may be elicited by pressure in the epigastric and dorsal regions.

Vomiting.—Vomiting is a much less constant symptom than pain. Here, again, much depends on the temperament and control of the individual. Sometimes it is distressing by its violence and constancy, at other times it is almost absent. But, to a certain degree, it is a very typical symptom of peptic ulcer. It is perhaps more constantly associated with ulcer inside the viscus than with duodenal ulceration.

It has several causes. Usually it is closely related to hyperæsthesia of the mucosa around the ulcer, but it may be the result of the retention of food in the stomach from interference with the pyloric function.

Most usually there is a close relationship between vomiting and pain. In those cases in which pain is not a prominent symptom vomiting may be largely in abeyance.

Vomiting of peptic ulcer has a definite relation to the act of ingestion. It occurs more quickly after food has been taken, when the ulcer is inside but is delayed proportionately in cases of ulcer outside the stomach proper (pyloric and duodenal). In the latter it is when the stomach begins to empty its contents, two to four hours after food, through the pylorus that vomiting occurs from the irritation and movements of the diseased area. This corresponds with an advanced stage of the digestive act, and when the acidity is at its highest. The patient commonly complains of the acidity or acridity of the vomited material, which may be so sour as to 'burn the throat' and set the teeth on edge.

Except in cases of retention, the quantity returned corresponds in amount to the meal taken.

It is little altered, and does not, as a rule, contain much mucus.

If the pylorus be occluded, with marked retention of the contents, the characters of the vomited material may differ greatly from the food ingested. It may then have the sour smell of fermentation, be frothy, and contain undigested particles of food and remnants of previous meals.

In all cases of peptic ulcer, blood, red or altered, in all proportions, may form a part of the vomit. If not discovered macroscopically it may be found as occult blood by testing.

The absence of vomiting should never exclude the diagnosis of gastric ulcer—especially of duodenal ulcer.

Hæmorrhage.—In some form and in some degree, bleeding is a very constant feature of peptic ulcer. In my own experience it occurs in about 80 per cent. of all cases. It is thus, next to pain, the most diagnostic feature of ulcer. It may be induced by the strain of vomiting or by the functional activity following a heavy meal, but it not uncommonly occurs when the stomach is quite empty and the patient at rest. It is thus a rather frequent occurrence at night.

The immediate cause of hæmorrhage is the opening of a blood vessel by necrosis or erosion. It occurs most frequently from an artery, but it may be venous or capillary.

Very commonly a severe hæmorrhage is immediately preceded by an exacerbation of gastric pain, which occasionally is agonising.

From time to time hæmorrhage may be the first sign of the existence of ulcer, although it more usually comes on after pain and other symptoms have existed for some time.

Hæmatemesis, or the vomiting of recognisable blood, occurs only in about 40 to 50 per cent. of cases of gastric ulcer. It is not nearly so frequent in duodenal ulcer. The appearance of the blood depends on the rapidity with which it is shed, the amount poured out, and the length of time it has lain in the stomach. It is brown (coffee grounds) or black when the amount shed does not produce rapid vomiting. It then lies in the stomach long enough to be changed by the action of the HCl, the gastric juice, and gases.

In most cases of hæmatemesis there is also melæna. It is not necessary that the stools, unless they contain an enormous quantity of blood, should have a tarry consistency and colour to recognise that they contain blood. Experience enables one, by the macroscopic appearance, to suspect the presence of even moderate amounts and verification may be had by testing.

That hæmorrhage should be such a constant accompaniment of ulceration of the stomach and duodenum is not surprising. It is a singularly vascular organ and has a rich blood supply from the three great branches of the cœlic axis. The anastomosis is very complete, so that a network of arteries spreads over the entire stomach, the larger branches of which can be seen through the peritoneum.

The situation and depth of the ulcer thus determine, in no small degree, the proclivity to hæmorrhage and its severity. The larger the hæmorrhage the more likely is it due to peptic ulcer. A hæmatemesis of three to four pints is almost always due to this

cause, and to an ulcer situated in the smaller curvature and the posterior wall of the stomach.

In such cases the patient may immediately die of syncope. Fortunately, even in severe bleedings, the issue is, as a rule, favourable; but the resulting anæmia is profound and the patient's convalescence slow.

It should be recognised that very copious hæmorrhage, even when not immediately fatal, may be the precursor of perforation. Small constant hæmorrhages, unassociated with vomiting, and thus often unrecognised, also induce, sooner or later, a very profound anæmia.

In duodenal ulcer, with only leakage of moderate amounts of blood, none may appear in the vomitus, and malæna may be absent. The stools and vomitus should, therefore, in all suspected cases of peptic ulcer be examined, at frequent intervals, for macroscopic and occult blood.

The intermittent appearance of blood, macroscopic or occult, in the stools, unaccompanied by vomiting or hæmatemesis, is suggestive of its origin in the duodenum or intestine; but the absence of macroscopic blood in the gastric contents and fæces should never make the physician exclude the possibility of ulcer.

In addition to pain, vomiting and hæmorrhage, which are by far the most constant and important symptoms, there are others which, though usual, are not always associated with peptic ulcer. Of these the more important are:—

(1) *Hyperchlorhydria or super-acidity.*—The chief functional derangements of this condition are a sense of fulness after food, heartburn, sour eructations, flatulent distension with belching of wind, sometimes pain, which may even be severe, and vomiting.

The appetite may remain normal, or be keen, although the patient may be afraid to eat. Sometimes it is perverted or capricious. The tongue may remain clean, and of normal appearance, or it may be too red and dry, or moist and furred. There is usually a pronounced thirst.

Hyperchlorhydria occurs in the majority of cases (from 80 to 90 per cent.) of peptic ulcer, but super-acidity, although the rule, is not absolutely diagnostic of ulcer. From time to time the acidity may be normal, and peptic ulcer cannot be excluded when hypochlorhydria or even achylia is present.

It must be observed that the symptoms of the functional secretory disorder known as super-acidity or hyperchlorhydria may be identical to those of ulcer. The physician will therefore not make the diagnosis of ulcer without confirmatory evidence (*see* Hyperchlorhydria.)

(2) *Gastro-succorrhœa*, hypersecretion or Reichmann's disease, is due to an abnormal action of the secretory glands, whereby large volumes of gastric juice are secreted into the empty stomach. The disorder may be (*a*) intermittent (gastro-succorrhœa periodica) or (*b*) continuous (gastro-succorrhœa continua chronica). The condition gives rise to similar, though more severe, symptoms to those which are associated with hyperchlorhydria. The symptoms are present mostly when the stomach is empty, and are therefore frequently nocturnal.

Gastro-succorrhœa may be present as a purely functional disorder, but when it is pronounced and persistent it may be regarded as almost diagnostic of duodenal ulcer.

Reichmann's disease should never be diagnosed from symptoms alone, but only on demonstration, by means of the stomach tube, of quantities of acid gastric juice in the fasting stomach.

The chief diagnostic points of peptic ulcer may be thus summarised: (i) Pain with definite relationship to the ingestion of food; with definite time of onset according to the situation of the ulcer; usually relieved by vomiting, alkalies, and sometimes by additional food, and occasionally by change of posture. (ii) Tenderness and muscular rigidity over some part of the gastric region—more commonly over the upper belly of the right rectus than elsewhere. (iii) Symptoms of hyperacidity—heartburn, waterbrash, flatulence. (iv) Hæmorrhage, sometimes macroscopic (hæmatemesis, melæna), more commonly occult. (v) The appearances obtained by X-ray and opaque meals.

Chemistry of Gastric Contents in Peptic Ulcer.—After the administration of a test supper and breakfast the gastric contents should be extracted, except in cases where the procedure is contra-indicated, and examined (1) quantitatively for free HCl and total acidity and (2) for occult blood. The prepared fæces must also be examined for occult blood by a proper and careful technique. A repeated positive reaction is of great diagnostic value. The significance of super-acidity and occult blood have already been referred to.

Prognosis and Treatment.

Prognosis of Peptic Ulcer.—Peptic ulcer must always be regarded as a grave disease. It is difficult to give a just estimate of its essential mortality, because so much depends on whether the case has been recognised and treated as ulcer at an early date. Under favourable circumstances the mortality, taken all over, is probably not more than 10 per cent. The main causes of death are hæmorrhage, perforation, and exhaustion.

If cases of peptic ulcer were recognised earlier and appropriate medical or surgical treatment adopted the mortality would probably be much reduced.

Treatment of Peptic Ulcer.—Although certain well-defined general principles may commonly be adopted, there should be no routine treatment of gastric ulcer. Each case must be dealt with according to its special requirements.

At the outset, before deciding on any plan of treatment, the most important consideration is that of hæmorrhage. If bleeding be profuse, all our energies must be directed to arrest it. When hæmorrhage is slight, treatment for the ulcer, without any special preliminary measures to deal with the bleeding, may be begun forthwith.

Severe hæmatemesis, however, requires immediate and careful treatment, often involving many procedures :—

(1) Absolute rest in bed is essential. Violent bleeding is always an alarming incident, and a varying degree of anxiety and excitement keeps the patient's circulation over-active. Everything should be done to reassure him, to calm excitement and quieten the circulation.

(2) Physiological rest for the stomach should, if possible, be attained. If the organ be kept quite empty and contracted, it is in the best position for the promotion of coagulation in the bleeding vessel.

There are other considerations, theoretic and practical, which may influence the physician to violate this principle and administer small quantities of egg albumen, or gelatine, even while bleeding is going on. When the distressing symptoms of marked hyperchlorhydria or super-secretion are present, the judicious administration of minute quantities of a bland and absorbing nutrient, like egg

albumen or gelatine, has much to commend it. Not only may local discomfort be relieved by the use of these remedies, but the patient's strength may be maintained. This subject will be more fully discussed in the general treatment of ulcer.

(3) *Medicinal Treatment of Hæmorrhage.*—The employment of medicaments for the arrest of bleeding has a very limited field. Most of the so-called astringents whose activity is dependent on tannic acid I have found worse than useless. Their introduction into the stomach during bleeding is apt to excite nausea and vomiting, and so aggravate rather than diminish the trouble.

There are, however, a few helpful remedies which have an influence in arresting or controlling hæmorrhage.

Ergot, though helpful in controlling certain forms of bleeding, is not universally useful. It may conceivably increase a hæmatemesis, arising from erosion of a larger arterial trunk, by raising the blood pressure. But ergot is generally very successful if employed for hæmorrhages from small vessels or from oozing surfaces. The drug has also a secondary beneficial action in promoting contraction of the gastric muscle. A reliable preparation should be used, and it is most active and rapid in its action when given hypodermically.

In most instances an opiate is called for. The best manner of administration is to give it by subcutaneous injection along with the ergot. Nothing answers better than one-sixth to one-fourth grain of morphine. It greatly assists in putting the patient mentally and physically at rest, and thus induces the most favourable conditions for the promotion of coagulation.

Many other remedies may also be employed for gastric hæmorrhage.

I believe I have seen good results from the oral administration of solution of adrenalin (1 : 1000) in doses of 15 to 20 drops. Its action is purely that of a local hæmostatic. The drug may be repeated every three to four hours.

Calcium chloride or lactate, in solution, may be administered in 15 to 30 grain doses per rectum. If the chloride be employed, it may be given in a few ounces of normal saline solution. It is also most efficient when given in 10 grain doses intramuscularly. If injected into the subcutaneous tissue it causes a good deal of pain.

I have found the following mixture, given by the mouth, in ℥ii doses every three hours, has an excellent controlling effect on most cases of hæmatemesis :—

Egg Albumen	1 oz.
Gelatin	2 drs.
Sol. Adrenalin	½ dr.
Calcium Lactate	1 dr.
Saccharine	2 grs.
Ess. Cinnamon	q.s.

This forms a jelly which can be slowly melted in the patient's mouth. It is best prepared by beating the egg albumen into a firm snow and mixing with it the melted gelatin, in which have been dissolved the other ingredients. It should be cooled rapidly on ice to prevent any separation and precipitation of the active materials. Its taste is not disagreeable, and can be varied by the flavouring chosen and by the amount of sweetening material incorporated.

Gelatin, administered both orally and rectally, I have found an excellent styptic and absorbent of excessive acid secretion. It should not merely be melted in hot water, but well boiled, so as to ensure thorough sterilisation. It has been found at times to contain the bacilli of tetanus.

If the hæmorrhage be severe we should not neglect to employ, either orally or intravenously, normal horse serum. It may be given in doses of ℥i or ℥ii. It is most efficient hypodermically. It may be repeated at intervals if necessary.

(4) Mechanical methods of treating hæmorrhage may supplement those given above.

The application of ice to the gastric region is a doubtful procedure. There can be little doubt that its application to the very vascular abdomen, by causing contraction of the vessels, will raise the general blood pressure, the very result we desire to prevent in all serious hæmorrhages. Similarly administration orally of ice is not to be commended. If, however, the ice-bag or the administration of ice chips is begun, it should be continued consecutively till bleeding has apparently been completely arrested. Its intermittent use is only a source of local vascular disturbance.

Another mechanical contrivance, which at least can do no harm and may sometimes have an auxiliary influence, is the ligature of

the thighs with an elastic bandage only sufficiently tight to obstruct the venous but not the arterial circulation. This causes a temporary reduction in the return of the blood, and may give time for coagulation to take place in the ruptured vessel.

After profuse hæmorrhage thirst is usually excessive, and the distress which it causes must be dealt with. It is impossible to comply with the patient's craving for large volumes of fluid. These would disturb the stomach locally and, when absorbed, raise the blood pressure and promote secondary hæmorrhage. The thirst is best dealt with by allowing the patient to frequently wash the mouth out with cold water and by the administration of rectal enemata containing gelatin and calcium chloride dissolved in three or four ounces of normal saline solution. By this means both the hæmorrhage and the resulting thirst are simultaneously treated. These enemata should be given not oftener than every four hours after recent hæmorrhages.

Syncope from gastric hæmorrhage should be dealt with by putting into immediate operation the general treatment of hæmatemesis; but, in addition, this special emergency will demand, without delay, further procedures. The lower end of the bed should be raised and, in very urgent cases, a warm sterile normal saline should be given intravenously. The amount should not be too large, so as to avoid excessive rise in the blood pressure. Simultaneously a warm rectal saline injection, with half an ounce of brandy, may be administered. Warm applications should be applied to the precordium and 1 cc. of Pituitrin administered hypodermically. No more than is absolutely necessary to arrest a fatal collapse should be done to raise the general blood pressure.

General Treatment of Peptic Ulcer.—When hæmorrhage has been controlled, or is only slight, general treatment may be begun. Peptic ulcer is still a medical ailment, and should be treated, in the first instance, by the physician.

Certain principles should be kept in view, and if possible attained in the treatment of peptic ulcer. Of these the more important are :

i. Physical and mental rest.
ii. The removal of all sources of irritation and infection from the broken surface.
iii. Physiological rest of the stomach.
iv. Dietetic and medicinal treatment.
v. Surgical treatment.

i. *Rest.*—Under the best conditions an ulcer may heal in about three weeks. Not less, therefore, than three weeks *absolute* rest in bed should be insisted on. The patient should be warned of the danger of indulging in violent movements or straining, and he should avoid, as far as possible, coughing. It is doubtful if it ever be justifiable to adopt an ambulatory treatment of gastric ulcer, even though the symptoms are not clamant.

Apart from the influence which physical rest exerts in promoting healing, it diminishes tissue waste, and thus assists the maintenance of strength on a very limited diet.

If necessary, mental tranquillity may be promoted by isolation of the patient, and under certain conditions by the judicious use of morphine.

ii. *The removal of irritation and infection from the broken surface* must be attempted by many procedures.

No solid food should be given, and such fluid nutrients as may be employed should be administered in strictly measured and limited quantities. In the early stages of treatment all distension of the stomach should be avoided, and in some cases it may be necessary to withhold even minute quantities of fluid.

The risk of superadded infection of the ulcer may be diminished by strict attention to oral cleanliness. The teeth should be brushed several times a day and an antiseptic mouth wash employed frequently, and all naso-pharangeal discharges dealt with. Certain antiseptic sedatives, administered orally, may assist in promoting these ends. (*See* Medicinal Treatment.)

iii. *Physiological rest of the stomach* is best obtained by keeping the organ as nearly empty as possible. When hæmorrhage is still active or recent, probably nothing but small doses of egg-albumen should be administered. Rectal alimentation by means of saline solution and glucose efficiently overcomes the thirst, although it is doubtful if it promotes perfect gastric rest.

If marked hyperchlorhydria or super-secretion be a feature of the case, it must be energetically dealt with and this source of irritation removed, which may be accomplished by diet and medicinal treatment.

iv. *Dietetic and medicinal treatment.*—In spite of the indication for physiological rest, even in cases of recent hæmorrhage, I am of opinion that the complete withdrawal of oral alimentation of all

kinds is a mistake. The maintenance of as good a general nutrition as possible is most important. The stomach is part of the body, and if food is withdrawn and nutrition fails, the vitality of the tissues is diminished as a whole and reparative processes retarded. Too often the dietary advised is nothing but a slow process of starvation, resulting after the ulcer has been healed in a profound debility, anæmia, and neurasthenia, requiring another and prolonged course of treatment.

There is one nutrient which fulfils many requirements and is at once a medicine and a food. Egg-albumin, administered in teaspoonful doses every two hours, has, in my experience, yielded the best results, even when hæmorrhage is going on. This nutrient is highly concentrated. It is very digestible and demulcent, and it has the capacity of absorbing or combining large quantities of free hydrochloric acid. In this way it diminishes gastric peristalsis and lessens the irritant and perhaps corrosive action of a too-acid gastric juice.

If there be no anxiety on account of hæmorrhage, a comparatively liberal dietary may be commenced at once. It should be fluid, bland, nutritious, and capable of absorbing a large amount of acid—in short, a food and a medicine combined. All foods which would mechanically excite the motor function of the stomach or increase its secretory activity are contra-indicated.

The dietaries best known are those of Lenhartz and Leube-Ziemssen.

Lenhartz Dietary.—This aims at maintaining the nutrition of the patient and thus promoting earlier healing of the ulcer. Much controversy has taken place as to the wisdom of oral alimentation in peptic ulcer with hæmatemesis. The choice therefore lies between starvation and the attempt to maintain nutrition by rectal alimentation. The latter method, as has already been pointed out, is open to many objections.

Briefly, this dietary consists of egg albumin with varying proportions of milk as treatment progresses. Small quantities of carbohydrate—sugar, rice, and rusk—are added from time to time; while, later on, raw meat and ham, finely minced, and butter are incorporated. Along with this diet, bismuth is administered in thirty grain doses, thrice daily. I have not found any real advantage in a strict adherence to the dietary as described by

Lenhartz. The most important items in it are the egg albumin and the milk. If these are given in proper proportions and in sufficient quantity, the essential requirements for the treatment of the early period are assured.

The Lenhartz treatment may thus be summarised :—

Absolute rest in bed is enjoined for three to four weeks. The patient is to be kept very quiet and all excitement avoided. An iced compress is applied to the gastric region to promote contraction of the stomach wall, diminish pain, prevent bleeding, and diminish gaseous distension.

With the food bismuth subnitrate is administered.

The entire egg is beaten up and slightly sweetened, and both it and the milk placed separately in glasses surrounded by broken ice.

The eggs and the milk are given alternately. The food allowed is administered in small quantities every hour. It has to be taken very slowly and thoroughly masticated. These conditions are fulfilled by never allowing the patient, for the first two weeks, to feed himself.

During the first week the eggs and the milk are increased daily till the patient is having 25 ounces of milk and six to eight eggs. For the second week no increase is made.

About the sixth day, one and a half ounces of finely scraped meat, incorporated in small doses with the eggs or given separately, is added to the diet. If this amount is well borne, it is doubled on the following day. At the same time some farinaceous food is begun in the form of softened zwieback (rusk) and well cooked rice.

At the end of the second week an ordinary light mixed diet is permitted. (See table for details of dietary.)

During the first week little detritus is left from the digestion of the limited diet, and the bowels are intentionally not moved to prevent disturbance from peristalsis. Later on (in the second week) glycerin injections are employed to promote evacuation.

Iron is administered orally to combat the post-hæmorrhagic anæmia.

According to Lenhartz, recurrent hæmorrhage, after his treatment, was reduced to 8 per cent.

Lenhartz's Dietary.

Day after Hæmorrhage.	Eggs.	Milk in c.c.	Sugar in gms.	Scraped Beef in gms.	Rice Pudding in gms.	Zwieback in gms.	Raw Ham in gms.	Butter in gms.
1	2	200	0	0	0	0	0	0
2	3	300	0	0	0	0	0	0
3	4	400	20	0	0	0	0	0
4	5	500	20	0	0	0	0	0
5	6	600	30	0	0	0	0	0
6	7	700	30	35	0	0	0	0
7	8	800	40	70	100	0	0	0
8	8	900	40	70	100	20 (1 piece)	0	0
9	8	1000	50	70	200	2 pieces	0	0
10	8	1000	50	70	200	2 ,,	50	20
11	8	1000	50	70	300	3 ,,	50	40
12	8	1000	50	70	300	3 ,,	50	40
13	8	1000	50	70	300	4 ,,	50	40
14	8	1000	50	70	300	5 ,,	50	40

For the first week the eggs are beaten and taken raw. During the second week one half of the eggs are cooked and one half are taken raw.

The success of any dietary is largely dependent on its palatableness. I have found that of Lenhartz distasteful to most English patients. It also lacks variety. For these reasons I have been in the habit of employing a dietary somewhat modified from the original described by Lenhartz.

In addition to rusk (zwieback) and rice, Benger's food, digested for one hour, can usefully be included. The butter may be spread on the rusks.

For the raw ham and minced meat, freshly prepared meat juice can be substituted without disadvantage. Each day's supply should be freshly prepared and kept chilled as described in Lenhartz's treatment. The best way to prepare meat juice is to take quarter of a pound of steak and shred it finely with fork and place it in a jar with two ounces of cold normal saline solution. Stir and press from time to time for two hours. Transfer the pulp to a stout linen cloth and firmly wring out all the juice. The amount obtained should be distributed among the egg feeds.

I have not found that there is any advantage in giving the egg and milk separately. The milk can be added to the beaten egg. When the milk gets much in excess of the egg I usually add ten grains of sodium citrate to the mixture to ensure that coarse coagulation of the milk will not take place.

The total quantities should be divided equally into two-hourly doses for the first two weeks, and for the third week into larger three-hourly doses. If the patient does not sleep well, two of these may be kept for administration overnight. If hæmorrhage be prominent, gelatin, in jelly form, may from the outset be added to the diet. The raw meat juice may be incorporated with the egg, or it may be given separately. The total quantity of Benger may be divided up into three equal parts, and be given at breakfast, lunch, and dinner hours.

Such a dietary I have found very well tolerated, and though not so liberal as that described by Lenhartz, it is safe, more agreeable, and suitable for almost every case. For the first three weeks nothing else should be permitted, unless small quantities of iced sterilised water.

It will be obvious that little debris will be left from the digestion of this diet, and if the bowels are moved once or twice during the first week, and thrice weekly thereafter, it will suffice. A small enema should be given or glycerin suppository used if evacuation is not spontaneous.

From the fifth day, and thereafter, daily, one c.c. of Molteni's* iron, arsenic, and strychnine, injected intramuscularly into the gluteal region, is given daily. This has an excellent action in aiding

* Molteni, Chemist, Florence, Italy.
Formula :—

Citrate of Iron	.05	in each ampoule of 1 c.c.
Sodium Arsenate	.001	
Strychnine	.001	

recuperation and overcoming anæmia without interfering with the stomach or causing constipation. I have found this preparation better than any other.

Bismuth carbonate, magnesium carbonate, and other neutralising drugs are serviceable during the period of treatment.

From the tenth day onward, if progress be good, a few of the eggs may be cooked to prevent the patient being nauseated by the monotony of his diet. Some of them may be scrambled with an equal quantity of milk, or they may be lightly boiled or made into custard pudding.

After-treatment of Ulcer.—On the completion of the third week a considerable alteration may be allowed in the dietary if the symptoms have subsided. The eggs, which are now becoming nauseous to the patient, may be gradually withdrawn, and some chicken and fish soufflé substituted. Minced tripe or ox-palate may also from time to time be added to the day's menu. The starches and sugar can be increased, and the meat juice withdrawn. Some toast, rusks or biscuit, with plenty of fresh butter, may also be taken, and the patient may gradually be put on to a light ordinary diet.

From the end of the third week I usually begin the administration of olive or cotton-seed oil. I have tried it earlier, but most frequently have found that it is not well tolerated. It is, however, one of the most valuable prophylactic agents we possess. It is nutrient, aperient, and a powerful depressant of super-secretion. It may be given in from one to four teaspoonfuls immediately before or after the three principal meals. Its taste, if a barrier to its use, may be disguised by half to one drop, to each dose, of one of the essential oils. For this purpose oil of peppermint answers well.

As a prophylactic measure against relapse an alkaline saline aperient is indicated. I have found nothing answer better than this formula :—

℞ Sodii Bicarbonatis
Sodii Phosphatis Exsiccati .... āā. ℥iss.
Sodii Sulphatis Exsiccati .... ℥i.
Saccharini .... gr. 5.
M.

Sig. :—℥i, in a tumblerful of hot water, one hour before breakfast.

This saline should be continued for a year or two after the ulcer is healed.

Leube-Ziemssen Cure.—A period of absolute rest, of three weeks, as in the Lenhartz treatment, is enjoined.

Nutrient enemata are relied on to sustain the patient if there be hæmorrhage. Warm applications are applied to the abdomen. After two or three days, or when the active bleeding is over, oral alimentation is begun. At first it chiefly consists of milk, barley-water, or thin gruel made from oat-flour (or sieved, if prepared from oatmeal), and some clear soup. Later on, eggs, soft-boiled or raw, chicken and sweetbread, creams, sago soufflé, and such like, are added. From these the patient is gradually promoted to ordinary light diet.

For the constipation which usually exists a teaspoonful of Carlsbad salts, dissolved in about half a pint of water, is ordered. Such an amount of fluid, even though only begun at the end of the first week, cannot, in my opinion, be administered without some risk of over-distension of the stomach, militating against gastric rest and producing a liability to secondary hæmorrhage.

Many other modifications of the Lenhartz and Leube-Ziemssen treatments are advocated by different authors, but none of them outstandingly offers sufficient advantage to necessitate detailed description.

Medicinal Treatment of Peptic Ulcer.—Medicinal treatment should, like dietetic measures, promote gastric rest, encourage healing, and mitigate pain or discomfort.

Bismuth preparations, especially the carbonate, given in doses of from fifteen to twenty grains every four to six hours, allay gastric irritation, protect the ulcerated surface, absorb hydrochloric acid, and are antiseptic.

Magnesium carbonate is also a preparation of great value in doses of fifteen to twenty grains alone, or, better, combined with bismuth.

Large doses of sodium bicarbonate should be discountenanced, even though the symptoms are relieved by this drug. Our aim should be not to get the gastric contents alkaline, but only neutral. Sodium bicarbonate, in large doses, after alkalising the gastric juice, only stimulates a fresh secretion of acid. This result can never be brought about by the employment of bismuth or magnesium preparations. Besides, sodium bicarbonate introduced into a

stomach containing much free hydrochloric acid evolves a large volume of CO_2, which may evoke hæmorrhage and pain by over-distending the stomach.

Cerium oxalate is also a useful remedy.

Belladonna and atropine are the best remedies we have for controlling hyper-secretion. No drugs are more useful for obtaining physiological rest to the stomach. Tincture of Belladonna, in five to ten minim doses, or half to one minim of the Liquor Atropinæ, combined with Bismuth and Magnesium, gives most excellent results. Such a combination checks both over-motility and hyper-secretion.

Some of the following prescriptions will be found suitable for the medicinal treatment of peptic ulcer :—

℞ Magnesii Carbonatis Pond :
Bismuthi Carbonatis .... aa. ℥vi.
Liquoris Atropinæ.... .... m. 12
Aquæ Chloroformi .... ad. ℥vi.
M.

Sig. :—℥ii, in water, thrice daily, after food.

℞ Cerii Oxalatis .... gr. v.
Magnesii Oxidi .... gr. xv.
Bismuthi Carbonatis .... gr. xv.
M.

Ft. Pulv. Mitte tales xii.
Sig. :—One powder every three or four hours.

℞ Calcii Carb. precip. .... gr. xv.
Magnes. Carb. Pond. .... gr. xv.
Bismuthi Subnitratis .... gr. xv.
M.

Ft. Pulv. Mitte tales xii.
Sig. :—One powder every three hours.

Lime salts, precipitated calcium carbonate, may also usefully be employed : and lime water, alone or mixed with milk, is beneficial. If diarrhœa be a complication they are particularly indicated.

Olive oil is indicated more in the after-treatment of ulcer and as a prophylactic than during the acute phase of the disease.

As has already been pointed out, in discussing the ætiology of ulcer, the protecting mucus is diminished or absent in very many cases. The condition of *amyxorrhœa* usually exists, and unless bleeding is severe it is best treated by passing into the stomach, through a soft tube, a 1 in 2,000 solution of *silver nitrate*. The whole procedure should be carried out with great care, and not more than half a pint introduced at a time. It should be allowed to remain for about one minute, and then be gently washed out.

Silver nitrate may also be usefully given, in pill form, by the mouth in gr. $\frac{1}{4}$-$\frac{1}{2}$ doses. It exerts a powerful antiseptic action. is distinctly astringent and hæmostatic, and promotes a copious mucous secretion.

V.—Surgical Treatment of Peptic Ulcer.—Peptic ulcer should still be regarded a medical ailment. Only under certain conditions may it become a surgical affection requiring operative interference.

It is highly important for the physician to recognise under what circumstances surgical aid should be invoked. A failure to appreciate when a peptic ulcer ceases to be a medical ailment very often compromises the life of a patient. The physician may fairly claim to treat with success the majority of peptic ulcers. Recent acute ulcers, and certain ulcers even of longer standing, if unassociated with stenosis at the outlet, serious mechanical deformity, or persistent hæmorrhage, may be entirely cured by medical treatment. All ulcers associated with obstruction at the outlet, from whatever cause arising, hour-glass deformity, or ulcers with recurrent or dangerous bleeding or persistent vomiting, and all those which have resisted prolonged careful medicinal and dietetic treatment, should be subjected to surgical procedures.

At the time of operation the appendix ought always to be removed, as it has important pathogenic influences in promoting peptic ulcer.

I see many cases, in which a gastro-enterostomy had been performed, seeking relief from the new symptoms which result from or follow the so-called curative operation.

A gastro-enterostomy undertaken in unsuitable cases is rarely permanently beneficial. The cases which undoubtedly do best are those with pronounced stenosis and retention, of not too long standing, in which the gastric motility is not seriously impaired. But when the outlet of the stomach is patent and emptying presents

no mechanical difficulty, operation should not be advised unless one of the conditions indicating surgical interference complicates the case.

Atonic dilatations and gastroptosis with a normal pylorus should not be subjected to gastro-enterostomy. If such stomachs cannot spontaneously empty through the normal pylorus, neither will they do so through the new opening which the surgeon makes. The stomach does not empty by drainage, but by contraction. If the motor function is absent, operative interference may only increase the difficulty by forming new points of fixation or introducing new symptoms.

Evolution and Complications.

Under the influence of rest and appropriate treatment an acute peptic ulcer may completely heal in about three weeks' time, and there may be no relapse. In forming a prognosis its duration and situation and the persistence of pain and hæmorrhage must be considered.

Once an ulcer has become chronic and the seat of infiltration and induration, relapses are very common, except under the most favourable conditions. Exacerbations and remissions characterise this condition. I have, however, had many cases of chronic peptic ulcer which have remained for years completely cured by strict adherence to a proper diet and regimen.

I. **Perforation of Stomach into the Peritoneal Cavity.**—This accident occurs in about 5 per cent. of all cases of ulcer and of those overtaken by the complication 80 per cent. die.

Perforation in young women under 35 occurs about three times as frequently as in men.

Ulcers on the anterior wall are more liable to perforate than those situated elsewhere.

This complication may occur during the course of acute and chronic peptic ulcers.

There may be premonitory symptoms, or the perforation may occur with dramatic suddenness.

If life is to be saved, it is absolutely necessary that this complication should be recognised at the earliest possible moment. Unfortunately it is a not uncommon experience to find that the symptoms significant of perforation are not grasped till general peritonitis has supervened and the patient is doomed.

The rapidity with which the symptoms develop, as well as their severity, largely depends upon the condition of the viscus when perforation occurs. If the stomach be quite empty, some time may elapse, after perforation, before acute disturbance occurs. In such cases there is not so much shock, and there is less likelihood of general peritonitis supervening. In all suspected cases of perforation the physician will remember it is imperative that nothing be introduced into the stomach, and that the patient be kept at absolute rest, preferably with the head of the bed raised on blocks and the head on a low pillow. This minimises the danger of the fluid collecting and remaining in the neighbourhood of the diaphragm and facilitates its gravitation to the pelvic cavity—a safer situation.

The perforation may temporarily close by the action of local peritonitis and the formation of adhesions. Immediate operation in such cases may be postponed till shock has passed and the general condition of the patient has improved. But these cases require, however, most careful watching, and unless the symptoms steadily and quickly subside, surgical treatment is imperative.

In the majority of cases where perforation occurs, when the stomach is full, the abdominal symptoms are more severe and the advent of peritonitis more rapid. The symptoms and signs, which should be recognised as characteristic of perforation, must always be present in the physician's mind in all cases of gastric ulcer.

1. *Pain.*—Its onset is sudden. It is frequently agonising, and is situated chiefly, at least at first, in the epigastric region.

2. There is marked *rigidity and tenderness* over the gastric region, particularly of the upper bellies of the recti. The patient lies in the dorsal decubitus, with the knees drawn up, to promote as much relaxation of the abdominal wall as possible and thus to mitigate pain. Pain, rigidity, and tenderness down the descending colon are rather characteristic of perforation.

3. *Distension* quickly begins in the epigastric region, and soon becomes general from the development of peritonitis, and the movements of the abdominal wall almost cease.

As soon as free gas accumulates in the peritoneal cavity, in any quantity, the area of hepatic dulness begins to diminish and is rapidly lost. The presence of free air in the abdominal cavity is of great diagnostic value in perforation of the hollow viscera. It can best be detected by auscultatory percussion. Normally,

when the stethoscope is placed over one pole of the abdomen and percussion made at the opposite pole, the sound is not well transmitted; but if free air be present in the peritoneal cavity the percussion is transmitted so clearly that it sounds as if it were made immediately under the stethoscope. Scratching, by means of a spicule of wood, at a point distant from the stethoscope, is even more delicate. The sound will appear, in the presence of free air, to be produced close to the stethoscope.

On auscultation friction sounds may be quite audible in various situations when general peritonitis is established, especially in the hepatic and splenic regions (perihepatitis and perisplenitis). The abdomen then becomes drum-like and the percussion note highly tympanitic. Later, the flanks may become dull from the accumulation of some exudate.

4. *Shock*, of varying degrees, is often a very early symptom of perforation. The complexion is ashy and the expression anxious. The pulse becomes thready, feeble, and rapid. The early pallor of the face may shortly be replaced by marked flushing, and an increase in the anxiousness of the expression (facies Hippocratica), when reaction takes place and the temperature begins to rise. A cold clammy perspiration bedews the body, and the temperature falls to a sub-normal figure. Later, during the stage of reaction, the temperature begins to rise, and may continue to do so till death takes place from the general peritonitis.

5. *Vomiting* occurs in the majority of cases, and even when the stomach is empty retching may continue. "Black vomit" is a grave symptom and persistent hiccough is of bad omen. Vomiting may be at times wholly absent.

6. The *respiration* is rapid and shallow, and thoracic in type.

Perforation of the full stomach, unless recognised within twelve hours of its occurrence, is well nigh hopeless. Often, as the general peritonitis develops, the patient ceases to complain of his pain, and the mitigation of the symptoms may lead the unwary physician to think his patient is improving. Before death, delirium and coma from toxic absorption are quite common, and life is rarely prolonged beyond four to seven days after the onset of general peritonitis.

II. **Perforation into various organs** may occur after perigastritic adhesions have been formed. In such cases the signs and symptoms may remain, at least for a considerable time, quite local and often rather obscure.

As ulcer is most commonly situated posteriorly on the smaller curvature, or in the duodenum, the organs most likely to become adherent to the stomach wall are the pancreas, liver, gall-bladder, the intestine, or the diaphragm. It is thus we may get a pancreato-gastric fistula, hepatic abscess, communications between the gall-bladder or intestine and the stomach, or subphrenic abscess and perforation into the pleural cavity or lung.

Of these complications *subphrenic abscess* is the most serious, and its early recognition is called for. Fortunately it is not a very common complication of peptic ulcer. So far as my own experience goes I have seen it occurring more frequently after rupture of an appendical abscess than from perforated gastric ulcer. Subphrenic abscess, owing to the symptoms and signs being indefinite, is unfortunately too often overlooked and may only be discovered at the autopsy.

The *rupture of a subphrenic abscess* follows the line of least resistance. Almost invariably the abscess resulting from a perforated peptic ulcer is situated in the left dome of the diaphragm or in the right hypochondriac region. The pus formation is, in the great majority of cases, intraperitoneal; but as the stomach is not wholly covered with peritoneum towards the cardia, where it lies in contact with the spleen, an extra-peritoneal abscess may form. The pus collection may develop rapidly or very slowly.

A subphrenic abscess may rupture into the pleural cavity or lung. A pulmonary abscess, with a surrounding septic pneumonia and necrosis, results, and ultimately a bronchus is perforated and foul-smelling blood-stained pus expectorated.

If a subphrenic abscess is not recognised and dealt with surgically, it usually ends fatally.

In all cases of peptic ulcer with severe pain and rigidity in the upper segment of the abdomen, associated with signs and symptoms suggestive of perigastritis or perforation, subphrenic abscess should be kept in view.

It should also be recalled that subphrenic abscess may develop, without actual perforation, from lymphatic absorption, and it should not be excluded because the more usual dramatic symptoms of its onset are in abeyance.

The diagnosis of this obscure condition rests on the following considerations :—

(1) The history of recent peptic ulcer with symptoms suggesting perigastritis or perforation.

(2) General symptoms and constitutional disturbance due to pus formation—*e.g.*, shivering, sweating, fever, and leucocystosis. When the abscess is well walled-in, a prolonged illness may ensue, with a hectic type of fever and much debility and emaciation.

(3) Abdominal symptoms and signs.—Of these, pain, fulness, tenderness, and rigidity in the upper segment of the abdomen are the principal. There usually is nausea, hiccough, and vomiting. The breathing becomes rapid and shallow, and the diaphragm is as much as possible immobilised, especially on the side on which the abscess is forming. Here much information may be got by investigation with X-ray, and screening gives the best results. The diaphragmatic movement may be accurately observed, and, when abscess attains a certain size, it may be seen that the diaphragm and heart are pushed well up into the left pleural cavity.

(4) Intrathoracic signs.—Most subphrenic abscesses, due to perforation of the stomach and communicating with that organ, contain air as well as pus. Many of the signs of pneumo-thorax are likely to be present. The percussion note over the base of the lung may be tympanitic, due to the air in the abscess cavity. The breathing may be amphoric and " metallic tinkling " heard. When the patient is ballotted from side to side, Hippocratic succussion may be audible. These phenomena are heard low down towards the base of the lung, and, at a higher level, there is an area of dulness. Later on, signs of pleurisy with pyothorax or pneumonic consolidation, ending in pulmonary abscess, may supervene.

III. **Perigastritic Adhesions.**—With every deep ulcer there is an involvement of the peritoneum in an inflammatory process. The perigastritis or periduodenitis causes adhesion of the parts to neighbouring organs or tissues. A conservative plastic operation, on Nature's part, is thus performed, which greatly diminishes the risk of perforation or limits its effects. The more extensive these adhesions become, the more likely are certain symptoms to be perpetuated long after the ulcer has healed. Indeed, permanent disability, due to mechanical interference with function, may result from these adhesions. Dense fibrous bands may contract and bind the stomach to neighbouring tissues and organs, and, if the adhesions involve the orifices of the stomach or the lumen of the bowel, signs of obstruction may develop.

The symptoms and signs of this condition are vague and inconstant. These are usually discomfort and dragging in the gastric region, when the stomach is full, and, sometimes, even pain coming on when peristalsis is most active. The symptoms occur usually periodically at a fixed time, and are in abeyance when the organ is empty and at physiological rest. X-ray of the opaque meal may aid diagnosis and reveal some alteration in the contour and peristalsis of the stomach.

IV. **Deformities** frequently follow peptic ulcer. Unless these are gross or involve the orifices, no symptoms or signs may be present; but any interference with either of the orifices at once produces characteristic and urgent symptoms.

When a peptic ulcer heals the scar tissue which results is endowed with the potentiality of progressive contraction. A considerable time may therefore elapse, after the healing of the ulcer, before the symptoms of deformity declare themselves. If the œsophageal or pyloric orifices are implicated in the scar tissue, signs and symptoms are not long in developing.

If contraction occurs at the cardia and involves the œsophageal orifice there will be some dysphagia and a tendency to regurgitation of food immediately after the act of deglutition. This condition may be confirmed by X-ray and by the use of the bougie or of the œsophagoscope.

Pyloric stenosis results from the contracting cicatrix, and the symptoms and signs increase progressively in severity and give rise to the characteristic picture of gastric retention (*see* Obstruction of Pylorus).

Scar tissue, resulting from ulcer anywhere between the cardia and pylorus, may produce a serious amount of deformity and contraction. If the contraction is extreme the stomach may be converted into two compartments, the so-called *hour-glass contraction*. The recognition of this condition is not always easy, and, as in the case of perigastric adhesions, the clinical symptoms and signs are vague and indefinite. Gastric discomfort, flatulent distension, and even pain may all be associated with this abnormality. A previous history of ulcer, or of acute gastritis with perigastritis, combined with persistent symptoms, may awaken the suspicion that hour-glass deformity exists.

The condition, however, can only be diagnosed by syphonage and by X-ray investigation with any degree of certainty. When the

stomach tube is passed, and apparently all the contents extracted, marked succussion may persist, which is very suggestive that fluid and air are being retained in a second compartment into which the tube has not penetrated. If the stomach be thoroughly washed out, and some time allowed to elapse, a second extraction may yield a further supply of gastric contents, which have regurgitated from the pyloric into the cardiac compartment. On this occasion the gastric contents may be bad-smelling and of low acidity.

Inflation of the stomach may also reveal the nature of the condition and show a notched outline which would be suggestive of this deformity ; *but X-ray and an opaque meal*, following a thorough lavage, will clear up the diagnosis. It is rather important to remember that a peristaltic wave may produce a distinct notch in the gastric outline on the plate negative, and lead the observer to a wrong conclusion. For this reason the fluorescent screen should be first employed and the stomach should be manipulated from the exterior so as to differentiate peristalsis from a permanent deformity. If the apparent deformity be due to peristaltic contraction, and not to a permanent abnormality, the manipulations will speedily alter the outline of the stomach and cause the notch to disappear or change its position.

V. **Chronic Indurated Peptic Ulcer is liable to become the seat of an engrafted cancer.** It has been said that every ulcer is potentially a cancer ; but this is only partially true. Scar tissue seems to be a favourable base on which a cancer may form. The growth begins almost always in the cicatrix or in the border of the ulcer. And yet in only about five per cent. of all cancers of the stomach can a diagnosis of previous ulcer be established. The development of an engrafted cancer on a chronic ulcer is very insidious, and its recognition is rarely early enough to permit of successful operation. For a time the symptoms and signs of ulcer mask those of the malignant disease. It is only by frequent examination of the gastric contents and by the gradual change in the clinical picture that the diagnosis may be made. If there be any suspicion, from the progress of the case, that carcinoma is developing exploration should be undertaken without delay.

While thus only a small proportion of cancers of the stomach appear to take their origin from a chronic ulcer the vast majority occurs in subjects whose previous digestive history has been unequivocally without flaw. (*See* Cancer of Stomach.)

The clinical picture of peptic ulcer is slowly replaced by signs and symptoms suggesting a change in the disease. The appetite fails, the bowels become constipated, the tongue gets coated, pallor and emaciation become progressive, and, later, well marked cachexia follows.

There may also be a corresponding change in the physical signs. There may now be added those of retention of the stomach contents and perhaps tangible tumour formation may be felt.

A slow but progressive change also takes place in the gastro-intestinal chemistry. From a hyperchlorhydria (HCl+), usually present in peptic ulcer, there is a slow and progressive diminution of free HCl till a hypochlorhydria (HCl−) and ultimately a total suppression of acid takes place. Lactic acid now replaces the normal acidity, Boas-Oppler bacilli are found in the vomitus or in the test breakfast, and occult blood becomes persistently found in the stools.

CHAPTER XIX.

NEOPLASMS OF THE STOMACH.

Tumours of the stomach are simple and malignant.

Simple Tumours are rare. They comprise lipoma, polypi, lymphadenoma, etc., but unless they occur at the pylorus they are of no importance to the physician. In this situation they produce all the signs of obstruction and require to be dealt with surgically. When situated in other parts of the stomach they are usually only recognised *post mortem.*

Malignant Growths.—Malignant growths are practically confined to the various types of carcinoma. The only other new growth of the stomach is sarcoma, and it is very uncommon. It may be primary or secondary. Primary lympho-sarcoma is most commonly met with. Its course is very rapid, and it is associated with pronounced metastasis. Its symptoms and gastric chemistry are those of cancer. It is therefore almost always diagnosed as carcinoma.

The differential diagnosis can be made if the supraclavicular glands become involved and one can be histologically examined. Enlargement of the spleen is absent in cancer but present in lympho-sarcoma. Metastasis in cancer is principally by the lymphatics, while in sarcoma it is chiefly by the blood stream. Sarcoma occurs earlier in life than carcinoma. According to Fenwick, sarcoma is more frequently associated with fever and albuminuria.

It should be noted by the physician that the absence of a visible or palpable tumour is no proof of the absence of malignant disease. When the neoplasm is at some distance from one or other of the orifices, especially when in the fundus, it may be impossible, by physical means, to diagnose its presence till it has assumed enormous size and projects under the ribs.

Cancer of the Stomach.—Carcinoma Ventriculi.

Incidence.—There is no organ of the body more frequently attacked than the stomach. It probably accounts for at least one-fourth of all the cancers. In this respect the uterus comes next to the stomach. My own experience leads me to believe that it is much on the increase, particularly in large cities. It may be alleged that better methods of diagnosis are responsible for a more easy and frequent recognition of the condition, and thus account for its apparent increase. Though this may be partially true, it cannot wholly explain the steady and progressive increase in the incidence of the disease.

From 30 to 40 per cent. of all cases are primary gastric carcinomas, and not less than one per cent. of all deaths are due to this cause. A decreasing mortality in tuberculosis and infectious diseases may be a factor in the apparent relative increase in the death roll of cancer.

It will thus be seen that gastric carcinoma is a common malady, occurring with alarming frequency. It is therefore of the utmost importance that the practitioner should be able to recognise it early when surgical treatment might possibly have the same chance of successful eradication as it has in mammary cancer.

Race.—So far as statistical data are available, there appears to be some immunity to the disease among the black races. In certain districts in South America and in Egypt it appears to be a rare disease. It should, however, be observed that among the negro races, Egyptians and South Americans probably a different incidence would have been found had adequate vital statistics hitherto existed.

Heredity.—Closely associated with race is heredity. It has been generally supposed that cancer is, like tuberculosis, to some extent hereditary. Whether this disease is really transmitted from one generation to another has not been definitely established. It is true, however, that gastric complaints tend to run in certain families, but my own analysis of 106 cases of gastric carcinoma tends strongly to disprove the influence of heredity in this disease. Not more than five per cent. of my cases gave a definite history of a family predisposition. It is thus evident that cancer of the stomach is, in the main, an acquired disease.

Sex.—Owing to the great frequency of uterine and mammary involvement, carcinoma generally has a larger incidence among

women than men. But so far as the stomach is concerned, the disease occurs more frequently in men. Of the 106 cases mentioned, 77 were in men. These figures correspond very closely to those of Osler, who found that out of 150 cases 126 occurred in men and 24 in women.

Age.—The age incidence is more important and striking. It is rare to have gastric carcinoma under the age of 30. It is conspicuously a disease of middle and advanced life. Seventy-five per cent. of all cases occur between the ages of 40 and 70. Quite exceptionally the disease has occurred in childhood, and the literature of this subject shows that such cases are pathological curiosities. It is thus apparent that carcinoma develops most frequently during the period of life when degenerative changes are in operation.

Etiology.—There is no subject in medicine more perplexing than the etiology of carcinoma. Its causation, in spite of world-wide and continuous research, remains wholly unknown. A study of the so-called predisposing factors leaves one quite unconvinced as to the real pathogenesis of the disease. Two conditions ostensibly predispose—*prolonged irritation* and *trauma*. When these exist, certain vascular changes become operative—hyperæmia, inflammation and hyperplasia—and conditions favourable to new tissue formation are present. Certainly there is little doubt that epithelioma follows or results from chronic irritation and trauma. This is well illustrated in carcinoma of the lip and tongue, apparently resulting from the prolonged irritation of smoking a short clay pipe or from the constant fretting of the tongue by a broken or rough tooth. But the same pathogenesis of carcinoma of the stomach cannot be so easily established. Chronic inflammatory changes in the gastric mucosa (chronic gastritis) might well be regarded as a provocative agent, and yet there is no reliable evidence that this form of irritation of the stomach wall produces cancer.

The constant ingestion of irritants, the bad habit of drinking fluids too hot, abuse of alcohol, and other irritating beverages, have been stated, but without demonstrable foundation, to be predisposing factors.

Malignant disease has an elective affinity for certain localities and tissues. In the stomach its proclivity to develop at, or near, the orifices is striking. It is by far more commonly found at the pylorus than elsewhere. Of gastric carcinomas 60 to 70 per cent.

are found in this situation. There must be some definite reason for the special liability of this part to become the seat of new growth. To what factor or factors may we ascribe this? Two at least are worthy of consideration. The orifices are functionally the most active parts of the stomach, and subjected to the greatest amount of strain and unrest. This is especially true of the pylorus and the pyloric region, and may be a predisposing factor in localising the disease in this situation. Again, the pylorus and its neighbourhood are very common sites of peptic ulcer and malignant disease may become engrafted on the scar tissue or on the edges of the chronic indurated ulcer. That there is some predisposition for scar tissue and chronic indurations to become the seat of malignant implantation appears to be established. Moynihan and Mayo Robson both claim that about 60 per cent. of cases occur in this manner, while William Mayo places the figure at 36 per cent.

But one is not wholly satisfied with this explanation. If it be true that gastric ulcer is more common in women, it is difficult to reconcile this fact with the higher incidence of carcinoma in men. It should be remembered, however, that gastric ulcer occurs in women at an earlier age, which is not so favourable to the development of carcinoma.

Besides, the truth is that clinically the vast majority of cancers of the stomach occur in those who previously have had entire immunity from gastric disorders.

The behaviour of carcinoma is in many respects suggestive of a germ disease. Certain organisms have been found, chiefly sporozoa. It has been suggested that these parasites excite cell proliferation. Later, and more complete, investigation, seems to show that these organisms are accidental and not pathogenic. The cancer cell itself behaves like a parasite, and the metastasis of cancer is a cellular transplantation which constantly reproduces the character of the parent growth.

In favour of the parasitic origin of cancer are some of the clinical features of the disease. Malignant growths in the stomach are prone to be associated with some fever suggesting the presence of toxins elaborated by organisms. The anæmia and the cachexia may have a similar origin. There are, however, other explanations for these symptoms, especially the auto-intoxication due to the decomposition in the alimentary tract from the altered chemistry associated with gastric carcinoma.

Again, carcinoma would appear to spread from one surface to another in apposition with it. It will also be observed that carcinoma primarily takes origin in organs and tissues which have a more or less direct connexion with the outside world. This suggests that an agent from the exterior excites the disease.

Virchow's connective tissue theory and Cohnheim's hypothesis of "cell-rests" are offered as explanations of the development of malignant disease in general. They do not, however, throw much light on the pathogenesis of cancer of the stomach.

Pathology.—The macroscopic appearances of gastric carcinoma are dependent on the type of cancer which forms the growth, on its stage of development, and on the secondary conditions which arise from its presence.

The important part of the growth takes origin in the epithelial cells, and most commonly from the cells of the glands.

There are four common varieties met with. In order of frequency these are:—(1) Epithelioma—cylindrical-celled adeno-carcinoma; (2) Medullary or Encephaloid carcinoma—soft cancer; (3) Scirrhous carcinoma—hard cancer; and (4) Colloid cancer.

To these might be added the less common extension of a primary squamous epithelioma of the gullet to the stomach.

It not infrequently happens that two types may co-exist, and it is often very difficult to sharply differentiate the more or less benign adenoma from adeno-carcinoma. Transitional forms are also met with.

Carcinoma rarely occurs as a secondary growth in the stomach, except as a metastasis from the mamma.

Characters of Different Types of Gastric Carcinoma.—(1) *Adeno-Carcinoma.* The new growth is rather soft, greyish-white in colour, of rapid growth and very vascular. It bleeds readily and is thus associated with frequent coffee-grounds vomiting. It sloughs and fungates easily. It forms the so-called "cauliflower excrescence," and has often a papillomatous appearance.

Microscopically masses of columnar cells are seen to project into the tubular spaces. At parts the lumen of the tubes becomes completely choked with cancer cells. Fibrous stroma is delicate, but abundant, and the connective tissue shows leucocytic infiltration. The growth tends rather to progress along the surface than to penetrate into the deeper layers of the stomach. The later stages of the growth are accompanied by interstitial hæmorrhages, followed

by necrosis and ulceration. Colloid degeneration is not an uncommon occurrence. The cylindrical celled cancer is most commonly situated in or near the pylorus. Metastasis is frequent.

(2) *Medullary or Encephaloid Cancers* are very soft, owing to the absence of extensive connective tissue formation. They are very vascular, and bleed freely. Ulceration is common, and frequently extensive. The general appearance of encephaloid carcinoma is not dissimilar to that of adeno-carcinomata. It occurs principally at the pyloric region, but it may involve other parts of the stomach. It spreads chiefly by the lymphatic stream, and many groups of glands become involved. Histologically the epithelial cells are smaller, more irregular, atypical, or embryonic than those of an adeno-carcinoma. The invasion by the new growth is very extensive, and all the coats of the stomach are, in time, implicated. Nests of cells in spindle formation are seen between the muscular fibres. Colloid degeneration or gelatinous transformation is of more frequent occurrence in medullary cancers than in adeno-carcinomata. The characteristic cancer cells are replaced by structureless jelly-like material, with only the debris of cells visible.

(3) *Scirrhus.*—Scirrhous carcinomata are characterised by their hardness, a feature due to the very abundant connective tissue formation. For these reasons this growth shows, in marked contrast to medullary cancer, little tendency to ulcerate till a late stage in its development. It is confined chiefly to the mucous layer. In this way it not infrequently happens that what appears to be a simple indurated chronic ulcer may really be a scirrhus.

This type of growth is almost invariably found to originate in the pylorus, which it may convert into a hard rigid tube with a very small tortuous lumen. The disease ultimately spreads from the pyloric region over the stomach walls—" cancer en nappe " of the French—and may convert the organ into a hard rigid contracted sac. The medullary type may be found to exist along with a scirrhus carcinoma. With hard cancer there is usually marked perigastritis with dense adhesions ; but from time to time a scirrhus at the pylorus may remain freely movable and suitable for radical operation. Scirrhus has less metastatic tendency than soft cancers.

(4) *Colloid Carcinomata* show degenerative changes of one of the above types. They are characterised by rapidity of growth and its great tendency to spread extensively to other organs and tissues. Colloid carcinoma is found more frequently in the abdomen than

elsewhere. A transverse section has a characteristic naked-eye appearance ; large spaces or alveoli are seen, filled with gelatinous material, which gives rise to the name " alveolar cancer."

Situation of the New Growth.—The analysis given by Welch of 1,300 cases of gastric carcinoma shows well the vulnerability displayed to this disease by the different regions of the stomach.

Pyloric Region	791
Lesser Curvature	148
Cardia	104
Posterior Wall	68
Whole or greater part of Stomach	61
Multiple Growths	45
Greater Curvature	34
Anterior Wall	30
Fundus	19

It will thus be seen that the vast majority of cancers have their origin in or near the pylorus.

Associated Changes in the Gastric Mucosa.—The gastric mucous membrane, in all cases of long-standing carcinoma, shows certain typical changes. These should be carefully remembered, as they account, at least to some extent, for many of the important clinical symptoms of the disease and for certain abnormalities in the gastric chemistry.

Generally the nutrition of the mucosa is interfered with, and there occur degenerative changes not dissimilar to those found in chronic atrophic and interstitial gastritis. Some observers maintain that these changes may precede the cancer and form a suitable soil for its development. The glandular and secreting tissues waste, and histological changes occur in the characters of the peptic glands. The very important alteration in the chemistry of the gastric juice—the disappearance of the free HCl—is probably due to these changes. As long as HCl is present and lactic acid absent, these histological changes will not be found.

Motor insufficiency is usually also a marked feature of gastric cancer, due to infiltration of the muscular tissue by the new growth. When stenosis at the pylorus occurs early, there may be a primary compensatory hypertrophy, with active peristalsis ; but sooner or later the motor function becomes seriously impaired.

Perforation of the stomach walls occurs in about six per cent. of all cases. As in simple peptic ulcer, the perforation may occur in a part which has become adherent to neighbouring tissues, and a local abscess may form, or the perforation may cause a leakage into the peritoneal cavity and general peritonitis. At other times the rupture may take place into the small or large bowel, or through the diaphragm into the pleural cavity, or pericardial sac, or into the abdominal cavity, producing carcinomatous peritonitis. The liver is very commonly the seat of secondary tumours from direct invasion.

The Symptoms and Signs of gastric carcinoma are usually very well marked, and not difficult to recognise. We are dealing with a disease which produces grave constitutional disorder, and according to its situation, pronounced local disturbances.

The constitutional symptoms, which may be described as the *cachexia* of cancer, are common to all cases, irrespective of its situation, but they are never more pronounced than in gastric carcinoma. The very serious disorder of digestion which results from the presence of a new growth in the stomach suffices to explain why the cachexia is more profound than that caused by the disease in many other organs.

At other times the symptomatology of gastric cancer is so indefinite and obscure that a correct diagnosis can only be arrived at by an exhaustive study of all the details of the case. It will make for accuracy in diagnosis if we consider (1) its general constitutional disturbance, (2) symptoms and signs of digestive derangements from a carcinoma in any situation of the stomach, and (3) the special symptoms and signs arising from the localisation of the new growth.

(1) **Constitutional Disturbance.**—General Symptomatology.

Progressive Debility, loss of flesh, anæmia, anorexia, perhaps some intermittent pyrexia, and constipation with occasional intercurrent diarrhœa, comprise the chief general symptoms. The general picture of gastric carcinoma may thus be summarised:

The *history*, *symptoms*, and *signs* of gastric carcinoma are fairly stereotyped. A man of some 50 to 60 years of age who has previously enjoyed excellent general and digestive health begins to lose weight and colour. His appetite, formerly good, progressively fails, and later on is lost altogether, or may even be replaced by a positive loathing for food. His bowels for the first

time in his life begin to give him trouble. He finds that he is becoming progressively more constipated, and has to rely on increasing doses of purgative medicine for relief. He becomes gradually weaker, and is easily tired and disinclined for work. His tongue becomes dirty and his breath bad. Nausea, more or less constant, with occasional vomiting, is added to his symptoms. Pain now becomes a usual feature of his case. The pain may be aggravated by the ingestion of food, but it is, unlike that associated with peptic ulcer, of a more persistent and indefinite character. Flatulent and fluid eructations are common. These eructations, or the vomit, are offensive in smell, and often very sour in taste. The vomitus may be discoloured from the presence of altered blood. As bleeding is usually small in amount, it takes the form of coffee grounds. It may be occult. The stools are frequently offensive, and may become darker in colour or black according to the proportion of blood which is present.

Every physician of experience is familiar with the difficulty of diagnosing, at an early stage, a gastric carcinoma which produces no pronounced symptoms or signs. When a new growth is situated in what we may call a ' silent area ' of the stomach, it may give no indication of its presence other than the constitutional disturbance. Such cases are usually overlooked and diagnosed, most frequently, as pernicious anæmia. The real condition is too often only revealed *post mortem.*

It thus becomes necessary, when cachexia develops somewhat suddenly in a patient between 45 and 60 who previously had been in good health, to study in detail all his symptoms and to differentiate them from those which may arise from diseases other than cancer.

Progressive weakness is not a symptom which is confined to gastric carcinoma. It may arise from such conditions as tuberculosis, diabetes, or pernicious anæmia ; but exhaustion is a very prominent feature of the malignant stomach.

Rapid and progressive *emaciation* is also decidedly typical of gastric carcinoma, especially when the growth is at the outlet. Not only is there the failure of nutrition from the arrest of digestion and the retention of the stomach contents, but there is the superadded toxæmia from the gastro-intestinal tract and from the growth itself. When patients are put on proper treatment there may be temporary improvement and increase of weight. The physician

should not let himself be deceived by such temporary amelioration of the symptoms, for relapse will soon take place. The cancer growth is probably associated with the formation of a protoplasmic poison which, when absorbed, exerts a catabolic action on the albuminous tissues and accounts for the excessive elimination of nitrogenous bodies in the urine.

Anorexia is more pronounced than in any other disease. Not only may there be a loss of appetite, but there is frequently a loathing for all solid food, and particularly for meat and fat. The loss of appetite is progressive, and is most pronounced when the motor function of the stomach is much impaired. In pernicious anæmia the appetite, in the early stages, is often increased.

Anæmia.—In no case of suspected carcinoma should an examination of the blood be omitted. This is specially true of those cases in which local gastric symptoms and signs are in abeyance and constitutional disturbance prominent. Cancers not interfering with either orifice or situated in the fundus may remain latent for long as nutrition is not seriously compromised. The true nature of such cases may frequently be recognised, and differentiated from pernicious anæmia, by a proper examination of the blood.

The degree of anæmia present in gastric carcinoma varies greatly in individual cases. The cachectic appearance, with the dirty yellow tint frequently seen in carcinoma, may make the physician think he is dealing with a more profound anæmia than really exists. While it is the rule that anæmia may be pronounced, a blood examination will show that the red blood corpuscles are rarely so reduced in number as in pernicious anæmia.

In gastric carcinoma the blood picture presents the type of a secondary anæmia with a low colour index. By exception, when marked stenosis at the pylorus with constant vomiting is present, the reduction of fluid absorbed into the blood stream may relatively increase the number of the red cell count beyond the normal figure. This polycythæmia is only apparent. That we are dealing only with a greater concentration of the blood (oligæmia sicca) can be demonstrated by the staining reactions and other microscopic features which will be found to present the characters of a secondary anæmia.

According to Osler, the average count of 59 cases gave :—Red cells, 3,712,186 per cubic millimeter, and megaloblastic formation was absent ; Hæmaglobin, 44.9 per cent.

There is usually some *leucocytosis* in gastric carcinoma. This is partly due to the recurrent hæmorrhages, complicating inflammatory changes and toxæmia. The increase in the white cells is present in rather more than 50 per cent. of the cases. The normal increase in the leucocytes following the digestion of a full meal is arrested in the majority of the cases of advanced gastric carcinoma. Myelocytes are found in the blood and, as the disease progresses, degenerative changes occur in the leucocytes. Neucleated red cells are sometimes met with, and eosinophilia is usually present. Thus the absence of the typical blood picture will prevent the physician from falling into the error of diagnosing pernicious anæmia.

When bloodlessness becomes profound, œdema of the feet and ankles is a common feature and, towards a fatal termination, general anasarca may supervene.

Pyrexia, which is an infrequent accompaniment of gastric carcinoma, is irregular, and as a rule not high. In these respects it is almost identical to the subfebrile movement commonly seen in pernicious anæmia.

The *urine* should be examined in all cases of suspected gastric carcinoma. The quantity is considerably reduced—due, no doubt, to diminished intake and to vomiting. The colour is high and the specific gravity raised. There is a tendency for the reaction to be less acid than normal. This is, however, not invariable. It is supposed to be related to the defective secretion of HCl. The nitrogenous elimination is commonly greatly increased in the form of uric acid derived from the excessive destruction of the albuminous tissue and the toxæmia. In estimating the nitrogenous output a comparison must always be made with its intake. The characteristic feature in gastric carcinoma is that the elimination is out of proportion to the intake. Owing to the diminished amount of food taken and the distaste for meat the total excretion of urea falls short of the normal daily excretion, while per ounce of urine it is considerably in excess. The chlorides are almost always greatly decreased, and urobilin is found in varying amount, as in pernicious anæmia, especially if the destruction of blood be rapid.

Other abnormal ingredients are usually present, especially in advanced cases. Acetone and indican are commonly found, while peptonuria may appear. The latter has been ascribed to absorption from an ulcerated surface of the stomach.

(2) **Symptoms and Signs of Digestive Derangement from a Carcinoma in any situation of the Stomach.**—In addition to all the preceding symptoms of general constitutional disturbance—debility, emaciation, anorexia, anæmia, certain changes in the urine, and perhaps some pyrexia—there are other symptoms and signs which are more directly referable to the digestive organs.

It is very necessary to obtain an accurate history of the clinical symptoms. In the vast majority of cases no evidence will be obtained of previous general or digestive ill-health. In a middle-aged man, whose previous digestive health was perfect, the sudden onset of dyspeptic symptoms with debility and loss of weight should always awaken in the mind of the physician the probability of carcinoma. By exception there may be a history of previous stomach trouble, with symptoms suggestive of gastritis or peptic ulcer. The relationship of peptic ulcer to carcinoma has already been referred to in the etiology of this disease.

Of the symptoms specially referable to the stomach a complaint of *heaviness, distension, and fulness* in the gastric region, coming on after every meal, is a very constant one. This sense of uneasiness and discomfort usually lasts as long as food remains in the stomach.

Constipation.—In a man who is up in years, and who all his previous life has had perfect regularity in the movement of his bowels, the advent of sudden constipation is often the earliest warning the physician may have of the development of carcinoma. In my 106 cases constipation was a marked and early feature in 87. In ten the bowels remained regular in their action, while in the remainder there was irregularity. A few had diarrhœa occurring intermittently. This intercurrent diarrhœa, coming on at varying intervals, is in all probability due to the irritation set up in the bowel by the stasis of its contents. Persistent diarrhœa is more a terminal feature.

Pain.—Pain is a very notable symptom, although in certain cases it is never complained of from the beginning to the end of the disease. Fortunately it generally comes on early and compels the patient to seek advice without undue delay. Osler reports that pain was a symptom in 130 of his 150 cases. Other writers put the figure even higher. It was a prominent symptom in 90 per cent. of my own cases.

Unlike the pain of gastric ulcer, it is more apt to be constant, without definite relationship to ingestion, although it may be aggravated by food. In those cases where gastric stasis is marked

and fermentation is active, the pain generally gets worse till the stomach empties itself. Pain which is not relieved by rest and diet is probably not due to peptic ulcer, but to malignant disease. In almost all the benign affections of the stomach a careful dietetic and medicinal treatment will speedily effect amelioration of the pain and other symptoms. When, therefore, in a few days no improvement follows such treatment, suspicion of carcinoma ought to be awakened.

The character of the pain is variable. It may be dull or very severe, gnawing or burning, and liable, exceptionally, to paroxysmal exacerbations. It is rarely so severe as in gastric ulcer.

With the pain there is tenderness to pressure, especially over the site of the tumour. It is generally in the epigastric region and posteriorly between the fifth and the twelfth dorsal vertebræ.

The relationship of the pain to the exact situation of the growth is worth studying. In cancer of the cardia the pain is most frequently referred to the left shoulder-blade, and when the growth is on the lesser curvature to the inter-scapular region. When the cardia is involved, the lower end of the gullet is not infrequently implicated, and in such cases the influence of deglutition on the pain should be carefully noted. If pain is evoked by swallowing, and is situated in the mid-sternal region, it is very suggestive of carcinoma of the cardia.

When, as is most usual, the pyloric region is involved and obstruction is present, the pain is then more periodic and has a definite relationship to the efforts of the stomach to empty itself. In most of these cases visible peristalsis will be seen during the height of the pain. If, and when, the tumour ulcerates, and the peritoneum becomes involved, the pain may become permanently greatly aggravated.

Nausea is not uncommonly a troublesome symptom, and contributes largely to the anorexia. It may antedate, or accompany, vomiting.

Hiccough and eructation of gas and fluid are associated mostly with pyloric stenosis.

Vomiting is a usual symptom of gastric carcinoma, although it is often a late feature. At times it may be absent throughout the whole course of the disease. The degree and frequency of vomiting are largely determined by the situation of the growth. In cancer

of the body of the stomach, away from the orifices, vomiting may be in abeyance. Naturally it is most persistent and severe when there is serious stenosis of the pylorus. When the cardia is involved and the œsophagus implicated in the growth there may be a great deal of regurgitation after ingestion from the food retained in the œsophageal dilatation above the site of obstruction. Even when the stomach is empty, vomiting of glary mucus and saliva may be persistent.

The characters of the vomited material.—The amount vomited is often very large when there is retention from pyloric obstruction. It may then not only come on after ingestion, but is specially liable to occur in the evening from the accumulation of the day's food. It is usually offensive in smell, from fermentation and decomposition. Mucus is present in abundance from an accompanying gastritis. In simple stenosis mucus is rarely present in quantity. If the vomitus be set aside to settle in a cylinder it will usually separate into three layers. At the bottom the solid debris settles, the middle fluid layer is muddy and turbid and of a tea-and-milk colour, while at the top there collects mucus and froth entangling in its meshes organisms and light flocculent material.

The hæmatemesis which occurs in cancer is usually small in amount. The blood is then readily changed and very frequently the vomitus presents the characteristic "coffee-grounds" appearance.

In early cancer there may be no bleeding at all till ulceration occurs. Oozing in minute quantities, from the vascular soft cancers, is a very common occurrence, even before ulceration takes place. Such minute hæmorrhages are apt to be entirely overlooked, because no coffee-grounds or melæna are seen. In my own series rather more than 50 per cent. had, from the onset of definite symptoms, either occult or macroscopic blood in the vomitus or fæces. In every case of suspected cancer occult blood ought to be searched for in the vomitus and prepared fæces.

The microscopic characters of the vomitus in cancer.—After the vomitus has stood for some time in the receiving vessel, a small amount should be taken from the bottom of the glass, by means of a pipette, and a drop placed on a microscopic slide. Another drop taken from the surface should be mixed with this and covered with cover-glass. The specimen should be examined with high and low power.

The microscopic characters of the vomitus will largely depend on the amount of gastric retention which is present. In those cases where the outlet is seriously implicated organisms of putrefaction and fermentation will be present. It will be noted that there is practically no digestion of flesh fibres. The transverse striæ are still seen, and the orange-red colour of the fibres still retained, showing that these structures have been practically unacted on.

Blood cells are frequently met with, and pus may be found when the growth has advanced to the ulcerative stage.

Starch granules are abundant and, when stained with a weak solution of iodine, show a variable and usually defective amount of digestion. Unaltered starch (dextrin) will be a blue-black, and partially digested (erythrodextrin) granules a burgundy colour.

Yeast cells are sometimes found when stagnation is pronounced. In contra-distinction to those found in benign stenosis, where they are mostly discrete, the yeast cells are frequently in chains. Various pyogenic organisms (staphylocci, streptococci, etc.) may also be present.

The most important and significant organism is the Oppler-Boas, or lactic acid bacillus. It is a large rod-like bacillus, 6 to 8 μ in length and about 1 μ in thickness. The more abundant their presence, the more likely to indicate carcinoma. These large organisms may occur in straight rods or be 'elbowed,' are non-motile, and may be discrete or in chains, while sometimes they tend to form a mycelial-like structure.

These organisms are anaerobic and gram-positive. Being hyaline in structure, they are best seen in a partially darkened field, and they show up well when stained with carbol-fuchsin or methylene-blue. The detection of these bacilli indicates the presence of lactic acid, but not necessarily of carcinoma. When, however, they are found in numbers, in an extracted test breakfast of oatmeal (Boas' test breakfast), their presence is of great significance. The observer will note that it is rare to find sacrinæ in the cancerous stomach. In the early stages of malignant infiltration of a benign stenosis sarcinæ may be found, but they rapidly diminish in numbers and ultimately disappear as the new growth progresses.

Occasionally a collection of cells or small particles of the growth are extracted with the test meal, or are vomited. It will always be very difficult to make a positive diagnosis from the cells, but if a small piece of the tissue be found it may at once afford evidence of

the malignant nature of the condition and sometimes of the locality of the growth. Recently, when withdrawing a test breakfast in a case with all the symptoms of carcinoma at the cardia, I found in the contents a portion of tissue which showed, on examination, that it was composed partially of squamous epithelioma, from the gullet, and adeno-carcinoma from the gastric wall.

Chemical characters of the Vomitus and Extracted Meal.—The vomitus will usually have a sour and offensive smell, due to lactic acid and decomposition products. No scientific data can be drawn from the chemical characters of the vomitus. The test breakfast, however, will yield reliable information. For the sake of accuracy, and when nothing contra-indicates it, the stomach should be washed out the night before, and a Boas' breakfast given the following morning. This lactic acid-free meal will enable the physician to exclude a fallacy.

The usual routine in the examination should be followed. Before retiring a dessert spoonful of currants should be swallowed, without much mastication, so that most of them will reach the stomach whole. The next morning, when the breakfast has been in the stomach for one hour, it should be extracted, filtered, and subjected to chemical examination.

(1) *Hydrochloric Acid.*—In the vast majority of cases HCl will be absent, or deficient in quantity. The more recent the case, the less likely is the acid to be seriously diminished, but with the progress of the disease there is a progressive reduction in the amount of free HCl till it ultimately disappears for good. Hence the importance of reviewing the gastric chemistry in a suspected case at not too long intervals.

By exception, in a very few cases, the free HCl appears to be present in normal amount and, as a rarity, there may even be, for a time, a hyperchlorhydria. A normal or excessive amount of hydrochloric acid is a misleading condition. It can sometimes be explained by the existence of a peptic ulcer in conjunction with a carcinoma, when the former condition seems to dominate the chemistry. The physician then will not be dogmatic as to the absence of cancer, in a case clinically suspicious, because he may at times find free hydrochloric acid in adequate amount. Nevertheless the rule holds good that in the vast majority of gastric carcinomas the free HCl quickly diminishes and ultimately disappears entirely from the gastric contents.

Again, free hydrochloric is commonly absent, as has already been indicated, in many conditions where no malignant disease of the stomach exists. It is almost always diminished or absent in chronic atrophic gastritis, in various forms of achylia gastrica, and in many constitutional conditions such as pernicious anæmia, cirrhosis of the liver, chronic parenchymatous nephritis, etc. Of more importance, according to Graham and others, is an increase in the mineral chlorides and a diminished ratio between the active acid and these chlorides.

(2) *Lactic Acid.*—When a hypochlorhydria is found, organic acid must always be sought for. Lactic acid is associated with the Oppler-Boas bacillus. If the precautions indicated above, in the administration of the test breakfast, are followed, the presence of lactic acid in quantity is as significant as the absence of free HCl, and almost diagnostic of gastric carcinoma. Lactic acid is also found in the rather uncommon syphilitic ulceration of the stomach. When free hydrochloric acid is absent and no lactic acid present, it is more in favour of simple achylia, although we cannot exclude cancer.

Lactic acid may be present in the gastric contents, when no malignant disease exists, in any condition associated with retention and stagnation. In such cases lactic acid is commonly associated with other organic acids (butyric acid).

The presence of a sufficient amount of HCl in the stomach contents inhibits the organisms which produce lactic acid fermentation, and when the latter is found in association with HCl it may be regarded as a contamination introduced with the diet or formed in the viscus by the decomposition of lactates. (For tests for lactic acid, see examination of test meal, page 124.)

Presence of Tumour.—It need hardly be said that when tumour can be detected we are dealing with advanced carcinoma. When the disease is not recognised till a tumour can be definitely located, it is usually too late to adopt more than palliative treatment. Unless malignant disease of the stomach can be strongly suspected or actually recognised before this gross sign is present, we must indeed confess that we have made little advance in the early recognition of this disease. If a careful and thorough examination of the patient has been made, the history and symptomatology of the case well considered and all the laboratory tests adopted,

we shall in the vast majority of cases be able to recognise cancer of the stomach before a tumour can be felt.

If the history, symptoms, and gastro-intestinal chemistry are against malignant disease, the possibility of an inflammatory tumour should not be overlooked.

In searching for tumour all the methods of physical examination should be carried out.

The tumour may be seen by inspection through an emaciated abdominal wall. Or it may be only recognisable during deep inspiration when it descends from the costal protection into the epigastric region. Palpation, with or without deep inspiration, should be adopted so as to determine, as far as possible, its relationships, movability and characters.

A neoplasm moves most freely when situated at the pyloric end of the stomach. Small tumours of the lesser curvature and cardia, and those on the posterior wall, are usually undetectable by the ordinary methods of physical diagnosis.

Sometimes a tumour of small size situated on the anterior wall or the greater curvature, which I have failed to detect by inspection and palpation, became at once demonstrable when the stomach was inflated. While the patient lies on his back the tumour is thrown upwards and outwards, and can be seen moving on deep inspiration, and readily felt by the palpating hand.

The tonicity of the stomach is much impaired in carcinoma, and evidence of marked ptosis may be present. In such cases a tumour at the pylorus may be overlooked, because it may be situated so low in the abdomen.

When the history and symptoms are all suggestive of carcinoma the development of a tumour is the most conclusive and diagnostic sign we have. It should be borne in mind that a ' tumour ' formation may result from simple peptic ulcer, especially when the ulceration has approached the peritoneum. Adhesions, thickened and inflammatory tissue, together form quite a large mass. I have frequently seen such masses at the pylorus. A careful study of these cases will usually clear up any doubt as to the nature of the tumour, but if any uncertainty remains, a laparotomy should always be advised.

No examination for a neoplasm of the stomach is complete without a careful examination of the umbilicus, the supraclavicular

glands, and the liver. Secondary metastatic invasion of the umbilicus is not infrequent in malignant disease of the stomach. The umbilicus becomes hard, distorted and indrawn. On palpation a cord-like feeling, passing into the deeper parts, may readily be felt.

Although all writers emphasize rightly the necessity of examining the supraclavicular glands, especially those on the left side, I have only in a small number of cases found them implicated. This sign is a late one as a rule, and is not so commonly met with as is supposed.

Radioscopy, when properly carried out, affords most valuable evidence of the presence or absence of carcinoma. In every doubtful case examination by X-rays should be undertaken. Often by this means alone, before symptoms or signs are more than suspicious, we may get early diagnostic evidence of the presence of a new growth. The wall of the stomach becomes deformed and its contour irregular. Barium, or bismuth, is frequently retained at the site of the growth, and an abnormal irregular outline results. Of course various deformities and simple stenosis will also produce aberrations in the contour and outline of the stomach wall.

Important further diagnostic evidence is to be found in the retention of the opaque meal for a period longer than normal. In simple achylia the stomach habitually empties precipitately. If, then, we have a clinical picture suggestive of carcinoma, and the absence of free HCl in the gastric content, over-retention of the opaque meal is strong presumptive evidence in favour of this diagnosis. But in cancer ' en nappe,' when the orifice is in no way interfered with, we may find the opaque meal leave the stomach comparatively quickly on account of pyloric insufficiency.

Specific Tests for Gastric Carcinoma.

Different observers have, from time to time, brought certain reactions forward as more or less diagnostic of carcinoma. Unfortunately up till now we have found no one test which can be relied on as definitely positive of cancer. Many of them are, however, when associated with clinical symptoms and certain signs and alterations in gastric chemistry, very helpful in difficult and doubtful cases. Some of them require an elaborate technique, and are therefore not so available to the practitioner. On the other

hand, some of them are quite simple. The better known ones will alone be described, and only those I have found of corroborative value in diagnosis will be given.

1. Wolff-Junghans Test (Albumin Test).—This reaction depends on the fact that no appreciable amount of soluble albumin is formed from food in the absence of HCl. If, then, in achylia an appreciable amount of albumin can be found in the *diluted* gastric filtrate, it is supposed to have come from the cancer 'juice' or secretion, and is regarded as positive of malignant disease of the stomach.

The gastric contents, after the test breakfast, must be carefully tested for occult blood. The presence of even a minute trace of blood would give rise to a positive reaction for albumin. As traces of blood are commonly present in all advanced cancers, the applicability of this test is thereby limited. But as this and other specific tests are employed principally to reveal *early* cancers, when hæmorrhage is less common and symptoms not conclusive, the drawback is more apparent than real.

In benign or simple achylia the amount of albumin found in attenuated solutions of the gastric filtrate is very minute, and this test aims at differentiating those from malign achylia.

Technique.—Take six test tubes of exactly the same size, and into each place measured quantities of gastric filtrate diluted to various definite strengths, with distilled water, so as to have the gastric filtrate diluted 1-10, 1-20, 1-40, 1-80, 1-100, and 1-200. The reagent employed is phosphotungstic acid in HCl and alcohol :—

Phosphotungstic Acid	0.3 parts.
Hydrochloric Acid	1 part.
Alcohol (90 per cent.)	20 parts.
Distilled Water	up to 200 parts.

One cubic centimeter of this reagent is floated, with the greatest care to prevent intermingling, on the top of the gastric filtrate. A milky ring (precipitated albumin) at the junction of the two fluids is a positive reaction.

When we are dealing with a simple or benign achylia, albumin is only got up to the dilution of about 1-20—*i.e.*, in the more concentrated solutions of the gastric juice. In malignant achylia the precipitate of albumin may be obtained up to 1 in 200, or even 1 in 400.

The method of carrying out the test and the conclusions to be drawn from the reactions will be seen at a glance from this table :—

1	2	3	4	5	6	Test Tube.
1 in 10	1 in 20	1 in 40	1 in 80	1 in 100	1 in 200	Gastric Filtrate.
W.M.	W.M.	W.M.	M.	T.	F.T.	Normal.
M.	T.	O.	O.	O.	O.	Benign Achylia.
W.M.	W.M.	W.M.	W.M.	W.M.	M.	Malignant Achylia.

W.M.=well marked. F.T.=faint trace.
M.=marked. O.=no precipitate.
T.=trace.

II. Glycyl-Tryptophan Test (Neubauer and Fischer).—This test is somewhat complicated, and certain fallacies which would vitiate it have to be eliminated.

While it is true that normal protein digestion is seriously interfered with in gastric carcinoma, owing to the absence of hydrochloric acid, it is claimed by Neubauer and Fischer that the carcinomatous new growth secretes an enzyme whose action is similar to that of trypsin, and that digestion of proteins may be by it carried further than normal. This ferment appears to have the power of hydrolising proteids and liberating amino-acids. Glycyl-tryptophan is the proteid employed. It is incubated for twenty-four hours with cancerous gastric juice, and tryptophan is split off by the tryptic ferment of the cancer. Glycyl-tryptophan is not capable of digestion by pepsin, and therefore normally no tryptophan is formed. When bromine vapour is added to the incubated gastric juice from the cancerous stomach, a reddish colour—the positive reaction—is struck in presence of tryptophan.

The test is carried out by following these details.

(i) The patient is well purged at the outset.

(ii) The following afternoon an Ewald's breakfast is given and extracted after being in the stomach for 45 minutes.

(iii) The extracted meal is then tested for bile, blood, and amylopsin, the presence of which would vitiate the test.

(iv) The gastric filtrate, in the absence of these vitiating substances, is treated with bromine vapour. A red colouration is positive of the presence of tryptophan.

(v) Before accepting a negative result as proof of the absence of tryptophan, 10 c.c. of the gastric filtrate are added to 2 c.c. of glycyl-tryptophan solution in a sterilised test tube, and a layer of toluol is floated on the top. The tube is then incubated at 37° C. for twenty-four hours. On the expiry of this period 3 c.c. are taken from the bottom of the tube by means of a graduated pipette, and placed in a test tube. Three or four drops of 3 per cent. solution of acetic acid are added, and bromine vapour blown slowly down the tube on to the incubated gastric filtrate. If no red colouration appears no tryptophan is present, and the test is negative of cancer. A positive reaction is indicated by the development of a colour which may vary from a pale pink to a deep violet red.

Fallacies and Precautions.—(i) Bile.—Any regurgitation of duodenal contents containing trypsin would, of course, vitiate the result by the formation of tryptophan in a normal stomach. Thus, if bile be present, duodenal regurgitation, with trypsin content, must be presupposed. Bile is best tested for by floating on to 5 c.c. of gastric filtrate, in test tube, a very weak alcoholic solution of iodine (of a dark sherry colour) and noting the presence or absence of a green disc at the junction of the fluids. If a green colouration fails to develop, bile is absent. Gmelin's test may also be employed.

(ii) Blood also contains a ferment which can split peptide and form tryptophan. The presence of occult blood must, therefore, first be excluded. Blood also appears to contain, especially during the digestive action, an antitryptic body which inhibits the action of the ferments absorbed and protects the tissues against their action.

(iii) Amylopsin may be detected or excluded by the hydrated starch test.

The originators of this test point out that the abundant presence of hydrochloric acid, 0.36 per cent., in the stomach will prevent a positive reaction. The test meal must therefore be quantitatively tested for free HCl. If it be found to exceed 0.36 per cent., another test meal must be given along with a neutralising alkali. For this purpose magnesium carbonate may be given along with the test breakfast in doses varying from 20 to 60 grains.

When the meal is drawn off it should be tested with congo red and blue litmus paper, and if found highly acid should be again neutralised by heavy magnesium carbonate or deci-normal sodium hydrate solution.

Although the persistent presence of sufficient quantities of free HCl is in itself presumptive evidence against gastric cancer it must be kept in mind that in early carcinoma the HCl content may be maintained for some time or even in the incipient stage of the disease be increased. Therefore in all cases where malignant disease exists this disturbing factor must be eliminated.

More recently Weinstein asserts that it is quite unnecessary to employ glycyl-tryptophan, but that we can test right away for tryptophan in the gastric filtrate. He admits only two disturbing elements, (a) the presence of free HCl in 0.36 per cent., and (*b*) duodenal regurgitation containing trypsin. He also believes that pepper and lemon juice interfere with the reaction, while sugar appears to favour the production of the cancer secretion containing the ferment.

Weinstein gives an ordinary mid-day dinner of meat, bread, butter, and milk, without any condiments. Plenty of sugar may be taken. Three to four hours later the meal is extracted, filtered, and examined for tryptophan. If found, the test is positive. Occult blood need not, he asserts, be tested for, as its presence does not vitiate the result. If it be accepted that blood contains not only a tryptic but also an antitryptic ferment, this view seems plausible.

It will thus be seen that there is considerable difference of opinion as to the reliability of the test, but I believe that, if carefully carried out, it affords some positive or negative evidence, although no absolute deduction can be formed from the result. It is only another observation which, in conjunction with the clinical picture and other tests, has some value. Further observation will alone determine its true value and how far we may rely on a positive reaction.

III. Salomon's Test.—This test consists in the discovery in the gastric contents of the albuminous secretion which comes from the surface of the neoplasm.

The day before the test is to be carried out, the stomach is thoroughly cleansed by giving the patient plenty of water to drink in the forenoon and, at 2 p.m., an albumin-free meal. To ensure thorough cleansing the stomach is washed out at night with 400 c.c. of warm normal saline solution. When the fluid has been removed it should be reintroduced so as to ensure thorough mixing with any contents which may have remained in the stomach. The total

albumin is then extracted by the Esbach's albuminometer, and the nitrogen by Kjeldahl's method.

Normally only a slight albuminous precipitate should be formed, and not more than 61 mg. of nitrogen in the 100 c.c. When more than 20 to 25 mg. of nitrogen are formed, and a well-marked precipitate of albumin (0.5 per 1000), the test is positive and cancer probable.

As negative results have been obtained in proved cases of carcinoma of the stomach, the test must be received with reservation. It is, however, rarely positive in simple conditions.

IV. Gluszinski's Test.—This test is only of value to differentiate simple peptic ulcer at the pylorus from malignant degeneration of the ulcer. According to Ewald it is only of use when peptic ulcer is certain and degeneration suspected.

The stomach contents are extracted three times in one day.

(1) The contents of the empty stomach.

(2) One hour after a test breakfast of egg albumin ; and

(3) Three or four hours after a meal of beef steak and potato purée.

If in one of these there is an absence or great deficiency of free hydrochloric acid it makes it probable that there is a malignant degeneration of the ulcer.

In cases where *no ulcer* is present, chronic atrophic gastritis (achylia) cannot be differentiated from malignant disease by means of this test. This renders it not reliable for primary malignant disease.

V. Abderhalden's Test.—This serum test is dependent on a general blood reaction, which probably only occurs in advanced cancers. When abnormal proteids find their way into the blood stream, reactive ferments are formed to promote their disintegration. Abderhalden's test for pregnancy is based on this supposition. In testing for cancer, pregnancy must be excluded.

The technique is as follows :—

A small piece of cancerous tissue is macerated, in a gauze sac, in a few c.c. of blood serum from the patient suspected to suffer from cancer. Later the tissue is transferred to a beaker containing a 2% solution of sodium fluoride, and kept at 22° C. for 36 hours. At the end of this time the fluid is tested for peptone. If peptone be found the test is positive. Although this reaction is promising it cannot, so far, be regarded as diagnostic.

VI. *Albuminose in the urine* has been found in 50 per cent. of cases of cancer by Aldor, so that when albuminosuria is persistent it is in favour of the diagnosis of malignant disease.

Special Features of Malignant Pyloric Stenosis.

A malignant stenosis causes very rapid deterioration in the patient's condition, not only on account of the malnutrition due to the mechanical obstruction, but also to the cachexia associated with the growth. It is rare for life to be prolonged beyond two years from the onset of the disease. On the other hand, a benign stenosis is compatible with life, and even a measure of health, for an indefinite period.

Pain in malignant obstruction is not only paroxysmal, but tends to be continuous ; while in simple stenosis it is chiefly confined to the digestive act.

Hæmatemesis in the form of ' coffee grounds ' is very habitual in malignant disease. Hæmorrhage is less common in simple stenosis, but if associated with ulceration it may occur and be more abundant and red in colour.

There is in all established cases of carcinoma a characteristic and diagnostic difference in the gastric chemistry.

In simple stenosis there is usually an increase of HCl. In malignant disease there is a progressive diminution, and ultimately total disappearance of the HCl acidity.

The total acidity in malignant disease is relatively high, and the disproportion between it and the free HCl is due to the excess of organic acid—especially of lactic acid.

Mucus.—There is usually an excess in carcinoma. In simple stenosis, unless associated with chronic gastritis, it is not increased.

The smell of the vomitus in simple stenosis is not abnormal, or it is that of fermentation, while in carcinoma it is usually offensive from putrefaction.

The microscopic examination of the vomitus, or test meal, is also very informing. The organisms found will depend largely on the presence or absence of a normal amount of free HCl. Thus in malignant disease the lactic acid bacillus abounds, and yeast may be present. If yeast is in chains, it is more characteristic of malignant than of simple stenosis. Yeast is not so commonly found in malignant as in simple stenosis, but in the latter the cells are more likely to be discrete.

Sarcinæ, in quantity, are practically confined to simple obstructions. *If very abundant, in a vomitus or test breakfast rich in HCl, they practically exclude malignant disease.*

My own experience shows this rule to be almost absolute. The analysis of the gastric contents from my last 233 cases of suspected ulcer, many of which had a clinical picture and history which was equivocal, confirms me in the belief that abundant sarcinæ in an acid gastric content (HCl) justifies the diagnosis of a simple lesion. This has been confirmed either through lapse of time or at the operation. In five cases only was there a departure from this rule, and in each of these there was a dual lesion—a cancer engrafted on the chronic ulcer. The possibility of this complication must, of course, be kept in mind. When it occurs, the gastric chemistry, in the early stage, is dominated by the presence of the peptic ulcer and masks that which is characteristic of malignant disease. In this way an error in diagnosis is easily made. In all cases which resist treatment and fail to progress, a frequent analysis of the gastric contents is called for. If malignant disease is becoming engrafted on a chronic ulcer, a steady and progressive decline in the HCl content will keep pace with the development of the carcinoma.

Occult blood, if persistently present, is more suggestive of malignant than of simple stenosis.

Prognosis in Benign and Malignant Obstruction.—Unless malignant stenosis can be diagnosed in its earliest stage, while yet a local disease, the ultimate prognosis is always unfavourable. Early operation has proved curative in a certain number of cases.

In simple stenosis the prognosis is excellent if the patient be subjected to operation before exhausted from starvation and auto-intoxication.

Treatment of Benign Obstructive Dilatation of the Stomach.—*Medical* treatment will only prove palliative if there be any serious degree of stenosis. Indeed, in some individuals, even a minor interference with the gastric outlet may produce severe symptoms. In others the symptoms may be slight and easily coped with when the obstruction is slight. The difference in the capacity of the stomach to deal with slight obstructive dilatation entirely depends on its motor efficiency. In all cases where stenosis is marked, no treatment will be permanently successful which does not remove the mechanical difficulty. The earlier the operation

is performed the more rapid and complete will be the recovery of the patient.

Palliative medical treatment may be tried in those cases where operation is refused, but only so long as it succeeds in maintaining the patient's strength and relieving his discomfort.

Briefly, it consists in lavage to prevent fermentation and auto-intoxication. It should be carried out at night and the stomach be thoroughly cleansed with some bland antiseptic solutions (*vide* gastric lavage, page 100), followed by sterile warm water.

The dietary should be fluid, and that recommended for gastric ulcer will be found suitable.

Medicinally, as hyperacidity is commonly present, bismuth, magnesium, calcium, and cerium salts are indicated. With these may be combined antiseptic remedies. Resorcin is particularly useful. Belladonna or atropine also allay gastric irritability and over-secretion.

When rectal alimentation has to be adopted it is fully time that the patient underwent operation. By this time his consent will be forthcoming, as his discomfort will have reached an almost unbearable point.

When the abdomen is opened the surgeon will best be able to determine which operation is indicated, but usually a posterior gastro-jujenostomy gives the best results.

Treatment of Malignant Stenosis.—Unless the disease is recognised very early, while yet the new growth is strictly intrinsic and complete resection can be accomplished, any treatment adopted can only be palliative. A gastro-enterostomy may give great relief for a time. With it may be combined certain medical measures which are described on page 260.

Cancer of the Cardia involving Œsophagus.—In this situation a cancer proclaims itself early, while a growth at the fundus may remain quiescent till very advanced.

Carcinoma of the cardia (which fortunately constitutes only about one-fifth of all gastric cancers) does not long remain without symptoms. Sooner or later the gullet becomes implicated and distressing and clamant symptoms and signs, of a local and general character, develop with great rapidity. The cachexia becomes profound, for the cumulative effects of slow starvation are added to those of the cancer.

From the progressive obstruction of the gullet, pain and difficulty in swallowing are increasingly complained of. The food is apt to be retained in or regurgitated from the dilated gullet. Pain becomes severe, and is situated in the lower sternal region and passes through to the back. X-ray and gentle palpation with a soft bougie will disclose the true nature of the condition.

Treatment of Cancer of the Stomach.

(I) Dietetic.—It will be obvious that alimentation will largely be controlled by the situation of the growth and the amount of interference with the orifices.

If the œsophagus is implicated in a cancer of the cardia there may be very strict limitations to the amount and kind of food which can be taken. The same is true in cases of malignant stenosis of the pylorus.

But when the mechanical conditions are more or less normal, as in cancers of the great curvature, the diet may be as varied and abundant as the patient can take.

We have to keep in mind that we are dealing with a disease which produces not only local but general disturbance. To overcome the debility and anæmia a nutritious and blood-making diet should be selected.

In neoplasms of the cardia with involvement of the gullet it may be impossible to get solid foods to pass through the obstruction, and only small doses of nutritious fluids, frequently repeated, may be available to maintain the nutrition. Ultimately obstruction may become so complete that even fluids cannot find their way to the stomach and starvation then faces the patient and hastens a fatal termination. In such cases rectal salines, hypodermic injections of olive oil, and the establishment of a gastric fistula may help to maintain life for a time and diminish the patient's distress.

When stenosis at the pylorus is present we are also faced with a great difficulty unless a gastro-enterostomy has been performed. In such cases, if no operation be undertaken, fluid nutrients, antiseptics, and digestive ferments may be of some use. Most relief, however, is obtained by daily gastric lavage, which prevents irritation, often stops vomiting, and relieves pain.

In all cases where it is possible to administer a meal I have found that the best results are obtained by giving six meals in the day at fixed intervals. Three of these should be nitrogenous, and three carbohydrate. The general absence of free HCl prevents peptic digestion of proteid aliments. This failure may be compensated to some extent by the administration of an antiseptic digestive after each nitrogenous meal. The carbohydrate and nitrogenous feeds should be given alternately, but no digestive administered after the starchy meals.

The principle of such a diet may be implemented as follows :—

8 a.m. *Breakfast.*—A concentrated meal in which nitrogenous material predominates is indicated. Raw meat pulp, beef juice, under-done mince and steak, mashed white fish, chicken and beef jelly, egg albumin, custard, curds and such like may be given in such quantities as the patient's condition indicates. The larger the amount of these materials which the patient can take the better will be his nutrition.

11 a.m. A carbohydrate diet of Benger's food (digested for one hour), various farinaceous gruels, biscuits, rusks, sugar and such like are aliments from which a selection can be made. Many of the other farinaceous foods on the market may find useful employment in framing a meal.

2 p.m. *Dinner.*—Nitrogenous food from similar articles as are enumerated for breakfast.

5 p.m. Farinaceous meal similar to that at 11 a.m.

8 p.m. *Supper.*—Similar to breakfast and dinner.

10 p.m. As at 11 a.m.

(II) Medicinal Treatment.—After each of the nitrogenous meals—breakfast, dinner, and supper—a dose of hydrochloric acid in mixture may be given. For example :—

R	Acidi Hydrochlorici Diluti	℥vi to ℥i.
	Glycerini Pepsini	℥ii.
	Resorcin	gr. 30
	Aquæ Chloroformi.... .... ad.	℥vi
		M.

Signa :—℥ii, in water, thrice daily after food.

As achylia is usually present, it should be noted that the dose of HCl must be large. The action of the free acid on the duodenal hormone (secretogen) and the formation of secretin may also help intestinal digestion by stimulating the pancreas and so preventing, to some extent, auto-intoxication from intestinal decomposition.

It is doubtful if the digestive activity of the mixture be greatly enhanced by the pepsin, but I have found that the glycerin is a useful addition. It is a good sweetening agent, is nutrient and antiseptic. The latter action is enhanced by small dose of resorcin and chloroform water, both of which tend to inhibit fermentation and decomposition in the stomach. Following the adoption of the treatment here outlined, I have seen such remarkable improvement in the general appearance and nutrition of the patient that doubt of the diagnosis of malignant disease arose. But such improvement is, of course, only temporary, and as the disease progresses the influence of treatment becomes a diminishing factor.

If anorexia be a marked feature, the appetite may be stimulated by the addition of bitters to the mixture. Of these the tinctures of gentian, nux vomica, condurango, and cinchona are most useful in appropriate doses. From a combination of nux vomica and condurango I have had excellent results.

Should pain be a prominent symptom it will be necessary to administer sedatives, of which opium or morphine are the most potent, either by adding them to the mixture or by giving morphine hypodermically.

Various other symptoms, such as constipation or vomiting, may require to be specially dealt with. Constipation is often most obstinate. It may be relieved by enemata or by aperients. Liquid paraffin and olive oil are often efficient and better than strong purgatives. At times, especially in advanced cases, diarrhœa may be troublesome and require treatment for its control.

Vomiting may demand the use of sedatives and lavage, unless the latter be contra-indicated.

The remedies which have been tried with a view to dissipating the neoplasm have not proved successful enough to be recommended.

Fibrolysin (Thiosinamine) has been used chiefly in carcinoma of the pylorus and œsophagus in the hope that it would relax the accompanying stenosis. It may be administered in half-grain doses in watery or weak alcoholic solutions, hypodermically, or it may

be given orally in one grain doses. I have not been able to convince myself that it has exerted any beneficial influence in obstructions of the pylorus or œsophagus.

The Trypsin treatment has likewise proved disappointing. In inoperable cancers it may be adopted for the sake of its mental effect on the patient, but there is no definite evidence that it has a restraining influence on the course of the disease.

The same remarks apply to the treatment by methylene blue.

(III) Mechanical Treatment.—The systematic use of lavage is indicated in all cases where stasis is present. It often gives marked relief from many of the most troublesome symptoms. The procedure should be carried out with care, and bland antiseptics may be used. If there is hæmatemesis it must be employed with the greatest care. A warm normal saline solution helps to remove excessive mucus from the stomach.

The application of warm fomentations to the gastric region may assist in relieving pain and help the patient to sleep.

Treatment by X-rays and radium may be tried, but in gastric carcinoma the results hitherto obtained have not been encouraging.*

Max Einhorn, in his most instructive and original work, 'The Duodenal Tube,' states that patients with cancerous stomachs can be better nourished through the duodenal tube than by any other method. The only limitation to the adoption of this treatment in all such cases is serious stenosis of the pylorus. There can be no doubt that the introduction of nutrient fluids, at the body temperature, directly into the duodenum, gives great relief to the stomach and prevents decomposition and fermentation, so that alimentary substances are fully utilised.

(IV) Surgical Interference.—If the disease is diagnosed early enough and the neoplasm is favourably situated, surgical extirpation should be attempted.

In malignant pyloric stenosis a gastro-enterostomy will prolong life when complete excision cannot be effected, and a gastrostomy may be carried out with the same object on cancerous stenosis at the cardia.

*For newer and more promising treatment of Carcinoma by X-rays see Chapter IX, page 97.

CHAPTER XX.

DEFORMITIES AND DISPLACEMENTS OF STOMACH.

True Obstructive Dilatation.

Many pathogenic factors are operative in the production of this condition. Of these the following are of most importance :—

(1) Cicatricial contraction from healed, or healing, peptic ulcer.

(2) Malignant disease invading the lumen of the pyloric-duodenal tube.

(3) Compression or constriction from tumours, inflammatory thickening and adhesions near to, but outside of, the pylorus.

Of these the more common examples are perigastric bands, enlargement of the gall bladder (cholelithiasis and cholecystitis) and enlarged glands (Hodgkin's disease and tuberculosis).

(4) Pylorospasm.

(5) Foreign bodies (hair-balls, etc.). Polypi.

The clinical picture of pyloric stenosis, no matter from what cause arising, is always characteristic. In addition to the classical symptoms referable to the mechanical obstruction at the outlet there are added others of a constitutional and local character in malignant stenosis.

Simple Pyloric Stenosis produces symptoms which vary in intensity according as the obstruction is slowly or rapidly developed. A certain amount of accommodation accompanies the obstructions, which are of slow formation, and the symptoms are not so urgent or dramatic. A very small amount of stenosis, if rapidly formed, suffices to evoke definite symptoms and calls early attention to the condition. It is thus that malignant disease in this situation never remains long latent.

Symptoms.—The appetite in simple stenosis is usually well maintained, and, indeed, owing to the malnutrition may be excessive until auto-intoxication from the absorption of the products of fermentation and decomposition sets in.

Similarly the tongue is at first clean, but may become later on thickly coated, especially in the morning.

Vomiting is usually an early and pronounced symptom. It is of the 'delayed' or 'collective' type. Thus it is most apt to take place at the end of the day, when a varying amount of remnants from all the meals collects in the stomach.

But the vomiting may take place after each meal, when obstruction is marked, and be of the 'ejaculatory' type. This is specially characteristic of congenital stenosis.

The amount thrown up is very variable. It may be enormous in quantity when the stomach is greatly dilated and the complete myasthenic stage reached. Thus each night, or every second night, pints of characteristic material may be vomited, containing not only the remnants of the day's dietary but that of previous days. Or vomiting may be less frequent still, and the stomach remain tolerant for several days or even weeks, when it may return with violence. These periods of remissions and exacerbations are familiar in all such cases.

When fermentation and decomposition are pronounced, much belching of wind after food is a constant complaint. The odour of the gas eructated may be diagnostic of the changes going on in the stomach. A well-marked odour of sulphuretted hydrogen is very characteristic of benign stenosis.

Pain in the gastric region is often a marked symptom. It may be paroxysmal, but usually is worst at or about the period when the stomach should empty itself. When very active visible peristalsis is present, the pain may be colicky or cramp-like in character. Evacuation of the stomach by the tube or by vomiting usually affords immediate relief.

The pain may not be confined to the gastric region, but may be referred to the pericardium and be anginoid in character; or it may extend to the shoulder blades or to the jaw.

In some cases the pain may never wholly subside between the exacerbations which are coincident with active peristalsis. This is due to the irritation and congestion set up by the prolonged irritant action of the retained and altered gastric contents on the mucosa.

The constitutional symptoms are principally those of malnutrition and auto-intoxication. The defective nutrition is seen in the emaciation, dryness, harshness and flaccidity of the skin, and dry

and lustreless hair. The diminished absorption of fluid, due to gastric retention and vomiting, is largely responsible for these alterations. It is also responsible for the scanty urination and the obstinate and pronounced constipation so usually present. The picture is indeed that of slow starvation. In all such cases acetonæmia occurs, and the breath is characteristic in odour.

Various symptoms of auto-intoxication are mingled with those of malnutrition. Acne, urticaria, and other vaso-motor affections of the skin are common, especially when butyric acid fermentation is present.

Nervous affections are usual developments. Headache, lassitude, insomnia are frequent; and tetany, though of comparatively rare occurrence, is usually of grave import. Vertigo is common. In young subjects epileptiform attacks have been noted.

Cardio-respiratory troubles are often a feature of this condition. There may be marked arhythmia, tachycardia, bradycardia, and other derangements of the heart's action. Attacks of so-called 'cardiac asthma' may occur, and if the patient has any tendency to true asthma attacks of this affection are apt to be evoked.

Most of these disturbances are of intermittent and irregular occurrence, and are not so common as might be expected.

Undoubtedly of these nervous affections *gastric tetany* is the most important. It tends to a fatal termination unless speedily relieved. The most tenable theory as to its pathogenesis is that of auto-intoxication. It has also been attributed to reflex irritation.

The spasms are usually confined to the hands and feet, and the flexor muscles are chiefly implicated. The contractions are both tonic and clonic, a feature characteristic of this disease. A spasm may be evoked by pressure or manipulation of the parts involved. There is an increased excitability of both motor and sensory nerves. Reflex response is abnormally active, and tapping and pressure over a nerve trunk produces hyperæsthesia in the area to which it is distributed.

Physical Signs of Obstructive Dilatation.—While the symptoms of this condition are pronounced, the physical signs are no less informing and characteristic. Naturally they vary in degree with the amount and nature of the obstruction. When it is of minor degree and compensation of the gastric muscle is adequate signs may be ill-defined.

It should be kept in view that dilatation, especially of the myasthenic type, may be associated with a general prolapse of the viscera (Glénard's disease). Such cases can usually be recognised by the presence of dropped kidney and by the displacement downwards of the upper border of the stomach.

The ordinary routine methods of physical examination should be applied for the detection of obstructive dilatation :—

On inspection, in the recumbent position, the abdomen is generally flat, from ill-filled intestines, except over the gastric region. Here some fulness may be detected, due mainly to gaseous distension of the stomach.

When the patient is examined in the erect posture the epigastric fulness will be seen to descend and now produce a distension in the umbilical region.

The most diagnostic evidence of obstructive dilatation is that of visible peristalsis. It occurs most frequently after food—most of all after a full solid meal ; but when stagnation is pronounced and the stomach never empty it may occur at intervals throughout the entire day.

Palpation confirms many of the signs observed on inspection. The gastric fulness is firm to the fingers, and the palpating hand can frequently detect alternating relaxation and contraction of the gastric wall and feel the peristaltic waves.

When obstruction at the outlet is suspected from the history and symptoms, it should not be regarded as absent because visible peristalsis is not observed at the time of examination. Many errors in diagnosis are thus made. As visible peristalsis is often only intermittently present, attempts to evoke it should be made. Stimulation of the epigastric region by flipping the skin with the fingers and briskly rubbing the lower costal and hypochondriac regions may bring about the contractions we are looking for. Visible peristalsis may also sometimes be evoked by placing a hand on either flank and shaking the patient from side to side, or it may be excited by ' dipping palpation ' over the stomach itself.

If these procedures fail, the passage of the stomach tube and gentle progressive inflation with a Higginson's syringe will almost certainly produce peristalsis if any serious degree of obstruction be present. Inflation will also define the position, outlines, and size of the stomach. Tumours situated on the anterior wall may

be projected forward and thus be more easily seen and felt, while neoplasms on the posterior wall will become less defined or disappear.

Tumours in the pyloric region should be sought for in the manner described in the chapter on physical examination (*q.v.*).

Palpation, properly carried out, will also produce splashing or succussion. When splashing is pronounced and easily elicited long after food, it is generally abnormal, and by locating the lowest point at which it is felt, the lower border of the stomach may be determined.

Percussion and auscultatory percussion are useful diagnostic methods which have already been discussed (*vide* page 84).

Most important information as to the amount of dilatation, motor function, degree of obstruction, retention, fermentation and decomposition can be obtained by a study of the test meal.

Lastly, X-ray examination will also give precise information as to the size, shape, and position of the stomach, and the degree and probable nature of the obstructive difficulty.

In this investigation I employ the following routine :—

(i) To ensure accuracy, the stomach should first be washed out at night.

(ii) About one hour later, the test supper should be given. It should consist of some light aliment, such as a rusk, with one dessertspoonful of currants swallowed without much chewing. This meal should be taken seven to eight hours before the test breakfast.

(iii) On the morning following the supper, the test breakfast of Ewald is given. It should be *extracted*, by means of Kuttner's extractor, as completely as possible, one hour after ingestion. The removal of the meal by simple expression is not satisfactory, as by this method it is difficult to make a true estimate of the amount in the stomach.

The removed meal should be studied macroscopically, chemically, and microscopically.

(1) *Macroscopic appearances.*—The amount extracted should be carefully measured. The size of the return will vary with the degree of dilatation and retention. If the maximum normal return be about 100 c.c., the degree of retention can be estimated by the amount this figure is exceeded. When we get 500 to 600 c.c., with a large waste residuum, we probably have severe obstructive dilatation with a varying amount of gastroptosis.

Next a portion of the meal should be set aside in a glass cylinder. In all cases of retention the meal separates into three layers. The lowest part is made up of sediment from the remnants of food (undigested starch, meat fibres, etc.). The middle part is watery and clear, or only slightly turbid, while the upper portion is usually mucoid, frothy, and muddy.

The odour, colour, and reaction to litmus and congo-red papers should be noted.

In benign stenosis putrefactive odour may be absent, but it is commonly present in malignant obstruction. During stagnation organic acids are formed in those cases where free HCl is defective or absent. Lactic and butyric acids may be detected by their characteristic odours.

The colour of the return has some importance. Blood, red or altered, will give a typical appearance to the meal. Dark grey or brown colouration, in the absence of free HCl, is commonly met with in malignant stenosis.

Note the presence or absence of currants. If no currants can be extracted from the stomach seven hours after the test supper, it is proof of the absence of any serious obstructive retention. On the other hand, if they are abundant, the evidence of a considerable stenosis is complete.

Note the presence or absence of mucus. In recent simple stenosis, without much fermentation or putrefaction, mucus may be absent; but in chronic cases with prolonged retention a catarrhal condition is set up and an abnormal amount of mucus will be found in the gastric contents. It is specially abundant in cancer.

Blood, red or altered to a brown or black colour, may be observed. Coffee grounds are typical of malignant disease, but blood so altered is not confined to cancerous obstruction.

(2) *Chemical Examination.*—Free HCl will be found in abundance in simple stenosis. It may be very excessive (hyperchlorhydria). The more persistently we have a normal or increased amount of free HCl, the more certainly is the case one of benign stenosis.

Conversely, the persistent absence of free HCl and the presence of lactic acid in quantity is strongly suggestive of malignant disease.

Occult blood must be sought for, and when HCl is deficient or absent the vomitus must always be examined for the various organic acids.

(3) *Microscopical Examination.*—It is well to examine, under the microscope, a drop from each of the layers as well as a drop of the mixed meal. It should be examined with high and low power, with and without staining by a weak solution of iodine. The degree to which stagnation and retention of the gastric contents is present will be at once observed by a study of the specimen. (*See* Examination of Gastric Contents, page 108.)

The microscopic appearances are indicative of a failure of digestive function. Meat fibres and starch cells will be found little altered. Often portions of food elements taken on a previous day can be detected. Sarcinæ are usually abundant, and yeast cells are commonly seen.

Hour-Glass Contraction of the Stomach.

This condition may be congenital or acquired.

It should, however, be remarked that the congenital type is not believed to exist by Mayo Robson and Moynihan.

The acquired form is most commonly due to cicatricial contraction following ulceration. It may also result from a diffuse cancer of the stomach wall (cancer en nappe).

The stomach becomes divided into two compartments of varying size and form. The fundal is usually larger than the pyloric sac. The condition may be complicated by adhesions of the stomach wall to the pancreas or neighbouring parts. Between the two compartments is a communicating canal of variable lumen. It may be so small as almost to amount to a complete occlusion.

Symptoms and Signs.—No definite clinical picture characterises this deformity.

The motor function of the stomach is, however, seriously compromised. Visible peristalsis of the upper sac may occasionally be seen.

Pain after food is a usual complaint, and there is very frequently vomiting.

The character of the vomitus is often the first thing to draw attention to the condition. At first it may be clear, but as retching continues it may suddenly change its characters and become turbid. The chemical reactions of the first and second portions of the vomited material may also be quite different. These variations are due to the contents of the lower regurgitating into the upper sac.

Emaciation in all these cases is generally pronounced.

Diagnosis.—The diagnosis of the condition can be made with most assurance by the use of the stomach tube and X-ray investigation.

(1) *Inflation.*—After the tube is passed the stomach should be inflated by means of a Higginson's syringe. Sometimes the irregular outline of the two cavities can be seen through the abdominal wall. This is especially the case if it happens that the viscus is depressed.

(2) When the stomach is *washed out* the retention of an abnormal amount of the fluid is very suggestive of hour-glass deformity (Wolfer's Sign).

(3) After apparently emptying the stomach, the persistence of succussion is also a suspicious sign (paradoxical dilatation).

(4) When, during lavage, the stomach has apparently been washed clean, the clear returning fluid is suddenly contaminated with debris, there is reason to think that we are dealing with hour-glass stomach. This occurrence is due to sudden regurgitation of the contents of the lower into the cleansed upper pouch (Wolfer's sign).

(5) The first part of the test meal, apparently completely extracted, may show signs of digestive failure, and may contain little or no free HCl. A little later, especially after changing the posture of the patient and manipulating the gastric region, a second and wholly different return may be obtained, showing a sufficiency of digestion and of free HCl. The first part of the test meal, that from the upper pouch, remained undigested and defective in free HCl because the duodenal hormone did not reach the cut-off fundal portion. The oxynitic (HCl) glands of that part of the stomach remained unstimulated and ceased to secrete.

(6) X-ray Investigation.—Even if the examination by the stomach tube leaves little doubt as to the nature of the condition, an X-ray investigation should never be omitted. The disposition and behaviour of the opaque meal will demonstrate the gastric deformity.

If the picture obtained on the screen be not quite definite, the gastric region should be manipulated to determine if we are dealing with a true deformity or only a passing local contraction. I have more than once seen a wrong interpretation put on such a spasmodic contraction, apparently dividing the stomach into two compartments, although no true hour-glass contraction existed. The possibility of this error must be carefully kept in mind.

An ulcer in the middle portion of the stomach may cause such an amount of local spasm that the stomach appears divided into two pouches before any cicatrisation has begun. Such a spasm can be dissipated by heat, atropine, or morphine, and by external manipulation. Observation in the dorsal decubitus will remove all doubt.

Another fallacy may arise from a gaseous distension of the colon, at the splenic flexure, pressing on the greater curvature and causing an indentation of the opaque meal. If the colon be cleared by a purgative or enema, or the gastric contents pushed upwards and the patient again screened, the apparent deformity will disappear.

In the *B.M.J.* of 1st January, 1921, there is an excellent contribution on Hour-Glass Stomach by C. Thurstan Holland, in which the whole subject is lucidly dealt with and a series of radiograms illustrating the condition given. This author's findings correspond almost entirely to my own observations, especially in regard to the relationship between simple ulcer and carcinoma. The summary of his paper is so informing that I cannot refrain from quoting it here :—

" 1. Hour-glass contraction from simple ulcer is a comparatively common condition—frequent in females, rare in males.

" 2. Its symptomatic history is a long one.

" 3. It is almost entirely an X-ray diagnosis.

" 4. It rarely ends in malignant disease—especially in females.

" 5. An hour-glass condition is found in a few cases, mostly in males, and with a short history, and which is caused by malignant disease. Its X-ray appearances generally differ considerably from those seen in the non-malignant type, and are suggestive.

" 6. The experiences of a large X-ray department are strongly opposed to the theory that it is usual for cancer of the stomach to be preceded by a simple ulcer."

Treatment.—We are dealing with a mechanical disability which can only be rectified by mechanical measures. Surgical interference is necessary, and when a free anastomosis between the two sacs is established rapid improvement usually follows.

Gastroptosis.

(*Synonyms* : Prolapse of Stomach, Enteroptosia, Visceroptosia, Gastroptosia, Splanchnoptosia, Glénard's Disease.)

These terms have been used loosely to define not only a downward displacement of the stomach, but prolapse of several of the abdominal viscera.

The amount of displacement of the stomach and of the other viscera varies greatly ; but when the stomach is much affected it is almost certain that conditions exist which make for a general displacement.

As so many terms descriptive of displacement of individual abdominal organs have been used, it will make for clearness if these are mentioned and shortly defined :—

Prolapse of the stomach—Gastroptosia.
,, Colon and Small Intestine—Coloptosia and Enteroptosia.
,, Kidney—Nephroptosia.
,, Spleen—Splenoptosia.
,, Liver—Hepatoptosia.
,, Heart—Cardioptosia.
,, Thoracic and Abdominal Viscera—Splanchnoptosia.

The term gastroptosia is now generally accepted as descriptive of prolapse of the stomach and of some other abdominal viscera. It is comparatively rare to find individual organs prolapsed.

In dilatation of the stomach it is chiefly the lower border which descends, but in gastroptosis the organ as a whole is prolapsed. The upper border also descends. We may certainly make a diagnosis of gastroptosis if, along with a descent of the smaller curvature, there is also a prolapse of the kidneys.

Gastroptosis is closely allied to gastric atony. It is also related to chronic gastric dilatation, which, in time, tends to produce visceroptosis.

Certain anatomical considerations should be kept in mind. The abdominal viscera are not held in fixed positions by attachments to osseous parts. These are all more or less movable. They are retained in situ (1) by the negative pressure of the cavity of the thorax, (2) by the mutual and interdependent support of neighbouring organs and fatty tissue, (3) by the parietes, and (4) by ligaments, blood vessels and nerves. The ligaments consist chiefly of folds of peritoneum.

Etiology.—There seems good reason to believe that gastroptosia is not merely a local defect, but is rather a congenital affection associated with a certain type of physique. Although the condition would appear to be acquired, the main pathogenesis is the congenital predisposition.

The build of those who may develop gastroptosis is characteristic. The over-tall, thin, light man with long neck, sloping shoulders, and flat chest is especially liable to the affection. Associated with these features there is usually a varying degree of weakness and relaxation of the abdominal wall which are potent factors in producing this condition.

A highly neurotic temperament is also an accompaniment of this type of ill-developed physique.

Too frequent pregnancies, tight lacing, lordosis, and certain thoracic affections (emphysema) and various intercurrent maladies associated with wasting (specific fevers, phthisis), want of exercise, fresh air, and suitable food may all become exciting causes of gastroptosis in those who have the predisposition.

Gastroptosia is a comparatively common malady among an urban population. It is far commoner in women than in men, and is almost always associated with movable kidneys.

Physical Signs of Gastroptosis.—The general methods already described for examination of the stomach should be applied to determine this condition. All the physical methods should be applied in the erect and recumbent postures.

By inspection in the recumbent posture the flat and relaxed abdominal wall will be noted, and often through it the visible pulsation of the abdominal aorta.

In this posture the ptosed stomach may resume more or less its normal position and, unless quite empty, give rise to fulness in the gastric and epigastric regions.

Palpation may reveal succussion, in an abnormally low position, long after the stomach should have emptied. The weakness of the abdominal wall, often associated with separation of the recti muscles, can likewise be felt.

Percussion and auscultatory percussion should be practised, and the outlines of the stomach mapped out.

Inflation is also a very informing method of investigation.

In the erect posture the stomach falls downwards, and on inspection the formerly full gastric region now becomes scaphoid and the umbilical and hypogastric regions show an abnormal fulness.

In pronounced cases percussion in the recumbent and erect postures will also give characteristic signs. If the stomach be empty a glass of water may be given to facilitate the examination.

In the recumbent posture the tympanitic note of the intestine may be elicited below the position of the lower border of the stomach, but when the patient assumes the erect posture the stomach is pulled downwards and a dull note replacing the intestinal tympanicity may often be made out right down to the pubic region.

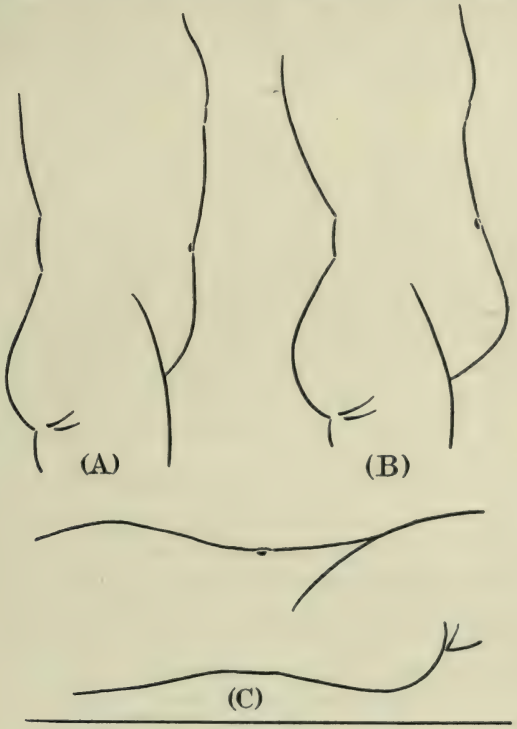


FIG. 30.
ABDOMINAL OUTLINES OF GASTROPTOSIS IN ERECT AND RECUMBENT POSTURES. Compare B and C with normal A.

When the empty stomach is inflated, it is usually easy to make out by auscultatory percussion the exact position of the organ.

Should any doubt as to the correct diagnosis remain, an X-ray investigation will eliminate all error. A plate should be taken in the recumbent as well as in the erect posture.

With a view to treatment, the stomach tube and test meals should be employed in all cases to determine the condition of the secretory and motor functions.

Symptoms may be almost entirely in abeyance in slight gastroptosis; but when the condition is advanced and pronounced, the amount of disturbance will largely depend on the patient's circumstances, environment, and ability to obtain proper treatment. When the sufferer from gastroptosis has to contend with adverse conditions, poverty, over-work, and unsuitable diet, and is obliged to maintain the erect posture for long periods, symptoms steadily increase in severity and end in chronic invalidism.

It will be obvious that the erect posture favours the retention of the contents, owing to the sagging downwards of the body of the stomach. It is thus that kinking arises, when the ptosis is extreme, and an actual, though temporary, obstruction is formed at the position where the duodenum is most closely attached to the spine; but under favourable conditions the motor function may be adequately retained and symptoms of stasis be entirely absent.

The Gastric Chemistry in Gastroptosis.—The gastric chemistry varies greatly in this condition. There may be hyper-, hypo-, or a- chlorhydria. It is of importance to determine the prevailing condition to initiate the best treatment.

Symptoms.—There is usually a sense of fulness and dragging in the gastric and epigastric regions after each meal. Sometimes there are also pains in the back and shoulders. These symptoms can be relieved by lying down, or, if the patient remains erect, by support of the lower part of the belly.

Nausea is common, and sometimes vomiting occurs; eructations of wind are frequent. Neurasthenic symptoms are nearly always present, which create a kind of vicious circle.

Prognosis.—When the disease is advanced, it is rarely curable, especially in those with the characteristic physique; but much can be done to ameliorate the trouble and to mitigate the most urgent symptoms.

Treatment.—In all young subjects who have the type of development which predisposes to gastroptosis, systematic physical culture should be insisted upon. All exercises which improve the chest capacity and strengthen the abdominal muscles will tend to prevent the development of the disease and prove the best prophylactic measures.

Improvement of the physique and general health must be one of our principal aims, and the neurasthenic and dyspeptic symptoms should be adequately dealt with.

In subjects who are too debilitated to undertake continued and systematic exercises, general massage should be advised, and while these measures are being carried out the patient should be kept in bed. Special exercises to develop the abdominal muscles should be devised to suit each case.

If deposition of fat in the abdomen can be procured, an increased intra-abdominal pressure will result, which will supply a useful support to the abdominal organs. With this end in view, rest in bed for eight weeks should be advised, and all other measures usually adopted in a complete 'rest cure' incorporated in the treatment. General massage should be carried out with olive oil and a liberal diet given to promote the formation of fat. Three large dry meals, at five hourly intervals, may be advised, and a pint of nutrient fluid should be very slowly sipped three hours after each of these. As long as the patient remains in bed, this method of feeding gives excellent results, and usually there is a rapid gain of weight.

Electro-therapeutic measures may also be invoked. High frequency and faradic currents, I believe, are sometimes useful.

There can be no doubt that the recumbent posture is im portant in aiding the ptosed stomach to regain some power, and mechanically it places it in the best position to empty efficiently and quickly.

As a further aid I am in the habit of making the patient rest with the lower end of the bed blocked up, to ensure that the stomach will be kept as high in the abdomen as possible.

Neurasthenic symptoms may further be relieved by suitable medicinal agents, which should be chosen to overcome atony and promote peristalsis. These pills will be found suitable :—

℞	Thyroidei Sicci	gr. $\frac{1}{8}$
	Strychninæ	gr. $\frac{1}{36}$
	Extracti Belladonnæ	gr. $\frac{1}{4}$
	Aloini	gr. $\frac{1}{6}$
		M.

Ft. Pil. Mitte tales 60.

Signa :—One pill thrice daily after food.

If prolapse and atony of the colon accompany the condition, these pills will also help to overcome the constipation.

Treatment of the dyspeptic symptoms will depend on the condition of the gastric secretion as ascertained by analysis of the test meal. Antacids will be indicated if hyperchlorhydria is found while hydrochloric acid should be administered if this acid is diminished or absent. (*See* Treatment of Hyperchlorhydria and Achylia Gastrica.)

Hypnotic suggestion may also be useful in promoting sleep and in overcoming the neurasthenic symptoms.

Several local treatments to establish support of the prolapsed organs may also be adopted.

If prolonged scientific exercises, along with the general treatment described, fail to bring about a sufficient improvement, we must then have recourse to mechanical abdominal supports. I am strongly of opinion that these should be a last and not a first measure of treatment.

It is useless to attempt to support an individual organ by means of pads. The various solid and air cushions commonly advocated for supporting a prolapsed kidney are worse than useless. They often only ensure that the displaced organ is retained in its abnormal position.

If abdominal support be considered essential, it is best accomplished by retracting the whole lower segment of the abdomen. For this purpose broad bands of leucoplast (zinc) may be employed, or the belts of Rose or Curtis may be found quite efficient. A well-made pair of short straight-fronted corsets will often supply the necessary support.

Surgical interference may be useful in exceptional cases; but for a general visceroptosis, due to congenital defect, nothing but disappointment will result from any attempt at fixation of an individual organ. If nephroptosis exists alone, a rare condition, and is demonstrably causing trouble, it is probably right to advise surgical treatment.

Congenital Stenosis of the Pylorus.

This condition is comparatively rare. It is probably due to general thickening, or hypertrophy, of the circular and longitudinal muscular fibres. Its exact pathology is still a debatable subject.

In some of the cases the symptoms are due to a pylorospasm, and not to a true stenosis. Such cases, as might indeed be expected, make a good recovery.

Very rarely pyloric stenosis of the congenital type has been noted in adults.

Symptoms and Signs.—Symptoms seldom come on immediately, but usually a few weeks after birth. The exact time of their advent will largely depend on the degree of stenosis present.

Ejaculatory vomiting, peevishness and crying, from pain, after food, constipation and unhealthy motions, visible peristalsis, detectable thickening or tumour at the pylorus, and progressive and rapid emaciation with all the symptoms of starvation, form the picture which characterises congenital pyloric stenosis.

The character of the vomiting is very suggestive. Immediately or shortly after each feed, some of the milk is suddenly thrown up (ejaculatory vomiting), no doubt due to spasmodic contraction of the stomach. The amount put up may be small or large. At times the quantity is so abundant that it obviously formed part of the last meal which has been retained.

Treatment.—It will be obvious that it is much better to restrict the amount of milk given at a time than to allow the hungry child to overload the stomach.

One of the features of this condition, to which attention should be drawn, is the remarkable temporary improvement which follows each change of food. We may take this as an indication for treatment, and make a change from one to another of the numerous infant foods at our disposal. Whichever food may be selected, it should always be given in small doses, and at longer intervals than those commonly prescribed in the early weeks of infancy.

Lavage should be tried ; it is often very efficacious.

Hot compresses applied over the gastric region appear to afford some relief, and minute doses of Dover's powder (gr. 1/24 to 1/12) mitigate the spasms. If the child is bottle-fed, a few grains of sodium citrate added to the milk prevents its clotting, and presumably makes its passage through the pylorus easier. Butter, or sour milk, sometimes agrees very well for a time, but whichever food is given with temporary improvement, too much optimism should not be indulged in that the crisis has been passed.

Nor has surgical interference been very encouraging. The mortality has been put as high as 60 to 70 per cent. after gastro-enterostomy ; but more recently better results have been reported in the journals. If, however, the case is resisting all forms of medical treatment, it is not fair to the surgeon to delay seeking his advice till the child is moribund.

It is a safe rule, whether we adhere to medical treatment or seek surgical aid, to give the parents a most guarded prognosis.

SECTION III.

FUNCTIONAL DISEASES OF THE STOMACH.

Under this head are included various disorders of secretory and motor function, nervous affections, dilatations, sensory disturbances and reflex dyspepsias.

It will be obvious that in many organic diseases we have accompanying disorders of both motor and secretory mechanism, and that no hard and fast demarcation can be made between functional and organic disease of the stomach.

Nevertheless a careful consideration of the history, symptoms, general condition of the patient, and a special examination of the gastric organs and functions will permit us to arrive at a sound diagnosis and differentiate the functional from the organic disease.

CHAPTER XXI.

DYNAMIC DISORDERS.

Disorders of Secretion.

While grave organic diseases, such as peptic ulcer and carcinoma of the stomach, are almost certainly associated with aberrations in the gastric secretion causing either an alteration in quality or in quantity, we may have similar changes brought about from purely nervous disturbances and from various errors in diet and regimen.

Aberrations in secretion, in quantity or quality, may be classified thus :—

(*a*) *Excess*—viz., Hyperchlorhydria and Gastrosuccorrhœa.

(*b*) *Diminution*—viz., Anacidity and Achlorhydria ; Subacidity or Hypochlorhydria ; Achylia.

(*c*) *Heterochylia.*

(*d*) *Diminution* or *excess* of *Mucus Secretion* { Amyxorrhœa, Myxorrhœa } Gastrica.

Excess of Secretion.

Hyperchlorhydria, or hyperacidity, while a usual accompaniment of peptic ulcer, occurs as a secretory neurosis, and may be brought about by indiscretions of diet. It may accompany supersecretion or gastro-succorrhœa and, as a rule, it is an antecedent of hyperchylia (gastro-succorrhœa).

By hyperchlorhydria is meant an increase in the free HCl of the gastric juice. Normally after a test breakfast the free hydrochloric acid should not exceed 0.2 per cent. When it persistently occurs much above this figure it constitutes hyperacidity.

It should be observed that true hyperchlorhydria results from digestive action, and that the symptoms correspond with the progress of digestion while hypersecretion (gastro-succorrhœa) may occur apart from ingestion.

When free HCl is excessive the total acidity will also be high. No inference should be drawn from the presence of high total acidity without reference to the amount of free HCl present, as the former may be due to an excess of organic acid. If free HCl is always first tested for, no error can be made in this respect. Indeed, when hyperchlorhydria exists, the presence of organic acids can be excluded.

When free HCl is persistently above 0.2 per cent., and when it reaches 0.3 per cent., I have most commonly found it associated with symptoms. On the other hand, the observation of the gastric juice of many young healthy subjects has convinced me that, by exception, a superacidity may exist without any symptoms to call attention to the condition.

No doubt nervous influences are largely responsible for the sensitiveness of the gastric mucuos membrane. A hyperæsthesia of the mucous membrane, if indeed such a condition exists, may account for those cases where a minor hyperacidity is associated with well-marked symptoms.

My own view is that pain, when present, is due rather to pyloric spasm and overactive peristalsis evoked by the supersecretion than to the direct action of the HCl on the mucous membrane.

At one time it was supposed that hyperchlorhydria might be due to over-concentration, and therefore diminution, in the amount of gastric secretion, but clinical experience proves that the reverse is more true and that almost all cases are associated with an excessive secretion.

After a test breakfast, extracted fractionally, it will be found, in hyperchlorhydria, that the free acid occurs earlier and more abundantly than in the normal subject. It may be found in quantity ten minutes after ingestion, and after thirty minutes it may be twice as abundant as normally.

The variations in quantity extracted from the stomach after a test breakfast depend on the amount of motility present. If hypermotility exists, and no pyloric difficulty be present, the stomach will empty very rapidly and only a small amount of hyperacid content can be extracted. Such a case would be diagnosed 'hyperchlorhydria.' On the other hand, if motility be poor, or obstruction at the outset be present from stenosis or spasm, an excessive return will be obtained and the condition recorded as 'gastro-succorrhœa.' Thus the amount of motility present plays a dominating rôle in these disorders.

Hyperchlorhydria is most commonly an ailment associated with youth and good health, and is specially liable to occur in the gouty and rheumatic. It should be regarded rather as a symptom than a disease, and diligent search made for its probable cause.

The ingestion of highly spiced and flavoured food, as eaten by the Anglo-Indian, excess of red flesh and extractives, too much salt, abuse of alcohol, tobacco, tea or coffee, and faulty mastication are all provocative of this condition.

The disorder occurs equally in both sexes. Various nervous disturbances seem to predispose to hyperchlorhydria. Thus it is common as a feature of emotional disturbances, grief, worry, over-work and hysteria.

Hyperchlorhydria may be a complicating feature of other diseases. It is usually present in chlorosis, in cholelithiasis, and renal-lithiasis, chronic appendicitis and, of course, it is a classical symptom of peptic ulcer.

Clinical Features.—Dyspeptic symptoms are usually gradual in their development and intimate in their relationship to the ingestion of food. A meal too rich in carbo-hydrates, too much starch and fat, is apt to evoke the symptoms.

The amount of disturbance varies greatly, from a mild discomfort to violent pain with sickness, eructations of wind and vomiting.

In mild cases there may be only discomfort coming on some time after ingestion and increasing as the digestive act progresses, to

reach its maximum about three hours after food, at the height of the acid tide. In more severe cases the symptoms are violent. These consist of a sense of pressure and fulness in the gastric region, with a hot burning feeling (heartburn) sometimes culminating in a gnawing pain. Throughout the attack the patient belches wind and frequently eructates sour mouthfuls of fluid. At times the patient may feel faint and look pale, and there may be anginoid pain and disordered action of the heart. The attack may pass off without further trouble at the end of the digestive process when the stomach spontaneously empties itself or when vomiting occurs.

I have never been able to verify the presence of visible peristalsis during an attack of simple hyperhydrochloria. In all cases where this has been undoubtedly seen, some degree of pyloric stenosis has been demonstrated to be present. But no doubt an excessive acidity may produce a degree of pylorospasm, and when a meal has been too large in amount and very badly masticated this may constitute a temporary obstruction augmenting the pain and producing a peristaltic colic.

During the attack there is usually tenderness in the epigastric region.

Most subjects of hyperchlorhydria have a keen appetite and rapid digestion, and are apt to eat too quickly and over-much.

It is not long before those who suffer from hyperchlorhydria learn that their symptoms are temporarily relieved by a meal rich in proteid element which has the power of combining large quantities of hydrochloric acid, and the habit of taking too much meat is thus readily acquired. But ultimately this only aggravates and prolongs the condition by tending to over-secretion—an excess of what Pavlov has described as 'adaptation juice.'

The tongue remains clean. Thirst is a frequent symptom, and constipation is the rule. Phosphaturia commonly occurs after each attack with a neutral urine. Constitutional disturbance is slight at first, but when the disease becomes confirmed some loss of weight may be noted. Headaches, mental depression, and insomnia are quite common.

It cannot fail to be noted that hyperchlorhydria in its clinical picture has many features in common with those of peptic ulcer. The test meal alone, in the absence of occult blood, cannot differentiate the two conditions; but the therapeutic test is important

and informing. Simple hyperchlorhydria readily yields to proper treatment, and the disorder may at least be kept in abeyance by continuous attention to diet and regimen with the aid of simple medicinal treatment. Peptic ulcer, on the other hand, requires much more drastic treatment before relief from the symptoms can be obtained.

Physical signs may be entirely absent, but diffuse tenderness over the gastric area is not at all uncommon. Some succussion may be elicited after ingestion, but in most instances it rapidly disappears unless the gastric contents are retained by obstruction at the outlet.

An examination of the gastric chemistry will reveal an excessive amount of free hydrochloric acid and a proportionately high total acidity.

In addition to the estimate of the free HCl in the extracted test breakfast, a microscopic examination of the sediment should be made. Stained, under cover-slip, with a very weak solution of iodine, a defective starch digestion can be demonstrated proportionate to the degree of hyperchlorhydria present. An excessive number of undigested starch granules (blue-black) will be found, and a large amount of erythrodextrin (burgundy-red granules) will be seen in the field. Little or no achroödextrin will be present.

Except in those cases of hyperchlorhydria associated with gastritis no excess of mucus will be seen in the extracted meal.

Gastro-succorrhœa, Hyperchylia, or Supersecretion (Reichmann's Disease).—As already explained, it is doubtful if this condition exists separately from hyperchlorhydria, and should not be regarded as a more advanced phase of that condition. But while the term hyperchlorhydria describes a secretory disorder confined solely to the digestive act, gastro-succorrhœa occurs apart from the ingestion of food and the digestive process. It is therefore convenient to describe it separately.

Clinically it is recognised by the enormous amount of the return when a test meal is extracted. Two causes alone can account for this : either supersecretion, which adds to the return, or retention of the meal in the stomach. Gastro-succorrhœa should not be diagnosed unless more than 200 to 250 c.c. can be extracted from the stomach after a dry test breakfast. For this purpose I usually employ two unsweetened rusks. These should be slowly masticated without any fluid. Indeed, before admitting the presence of a true

gastro succorrhœa, I prefer the more valid evidence of an abundant secretion in the fasting stomach. If the stomach tube be passed in the early morning, before any food has been taken, and more than 20 to 30 c.c. of fluid can be extracted, this may be regarded as proof of supersecretion. But even when these conditions are carefully fulfilled, such cases ought not to be too readily ascribed to gastro-succorrhœa. Further investigation will sometimes demonstrate that the excessive amount found in the stomach is nothing but a collection of normal gastric secretion which the myasthenic stomach has failed to pass on.

It is, in my opinion, very doubtful if a true gastro-succorrhœa ever occurs as a neurosis apart from pathological change in the gastric mucosa. It will be found almost always due to duodenal ulcer, or to the so-called acid gastritis of Boas.

It should, however, be noted that Strauss regards gastro-succorrhœa as a disease which can exist without myasthenia gastrica or retention from interference with the outlet. He argues that even when the stomach is kept quite empty, and nutrition alone maintained by nutrient enemata, an excessive amount of gastric secretion can be removed from the fasting stomach. This argument is not, however, convincing, because it is well known that rectal enemata excite gastric function.

Strauss believes that the disease is due to an excessive irritability of the secretory mechanism.

According to its severity and degree of development, this disorder is thus classified :—

(*a*) *Gastro-succorrhœa Continua Periodica.*—This constitutes a relapsing or intermittent form of the disorder.

(*b*) *Gastro-succorrhœa Continua Chronica.*—In this form we have a more or less continuous over-secretion.

The clinical symptoms of these conditions are those of hyperchlorhydria of the severe type, and it ought to be borne in mind that the more persistent the symptoms the more certainly are they the result of organic disease of the stomach. *In every case duodenal ulcer should be kept in view.* It is in those cases of gastro-succorrhœa chronica that nocturnal hunger-pain is often met with, and the symptoms most commonly occur in the small hours of the morning. The contents of the stomach and the prepared fæces should be examined repeatedly for occult blood, and an opaque meal given, and the conditions noted by means of X-rays.

Treatment.—Hyperchlorhydria and gastro-succorrhœa may be considered as one condition so far as treatment is concerned. In all persistent and intractable cases it is always safer to regard the condition as due to peptic ulcer, and to adopt the treatment prescribed for that disease.

On the other hand, when the attacks are infrequent and passing, very simple treatment may suffice to remove the symptoms. A few days very light fluid or semi-fluid diet and some alkaline mixture may be all that are required.

For the more severe and persistent cases a prolonged course of prophylactic dietetic and medicinal treatment should be instituted. Preventive treatment is of far greater importance than the cure of the established disease.

Food should be masticated well, and should be light and easy of digestion. All cured and salted flesh should be forbidden. No condiments should be allowed, and tobacco, tea, coffee, and alcohol must be used with great frugality. Good hygienic surroundings are equally essential, and fresh air and exercise should be insisted on. Physical culture at home, general massage, and hydrotherapy I have found to be most beneficial. A quiet life and freedom from excitement and over-work should be obtained as far as possible. Constipation should be dealt with by means of a morning saline or other simple purgative.

Dietetic Treatment.—The reader is referred to the treatment described for peptic ulcer. Either this should be carried out in its entirety or, if the case be less urgent, in a modified form.

Egg-flip, milk and lime water, well-digested Benger's food and jellies may be given at regular intervals and in sufficient quantities to maintain the nutrition of the patient.

Medicinal Treatment.—The same drugs as are employed for the treatment of peptic ulcer are useful for the relief of the symptoms of gastro-succorrhœa. Bismuth, magnesium, cerium and lime salts, in appropriate doses, may be employed alone or in combination. With these should be combined tincture of belladonna or atropine, very powerful depressors of secretion, in doses short of producing disagreeable physiological action. Olive oil, in one to two drachm doses before food, is an excellent remedy, nutrient and aperient, and I have had the most satisfactory results from its use.

A few weeks' course of gentle lavage of the stomach, in the early morning, can also be strongly recommended. The first pint of

sterilised warm water should be syphoned off, and a pint of a 1 in 2,000 to 4,000 solution of silver nitrate slowly introduced. This should be allowed to remain in the viscus for about a minute, and then likewise syphoned off. This solution tends to promote a myxorrhœa, and the mucus so poured out is protective to the gastric mucous membrane. It also exerts an inhibitory influence on the gastric secretion.

If pain be present during the attack, warm applications are soothing, but if not sufficient to allay pain, opiates may be employed in small doses.

Lastly, if a few weeks of thorough treatment fail to bring any durable relief, surgical interference may require to be considered. Such cases are usually symptomatic of chronic ulcer.

Diminution of Gastric Secretion.

A very large experience of the analysis of gastric contents confirms me in the opinion that conditions of subacidity, anacidity, or achylia are very common, either as permanent or passing affections. Hypochlorhydria in the young is usually evanescent. Subacidity or hypochlorhydria may follow some devitalising illness, and persist till the general health is restored. When this condition resists proper treatment and is permanent, it most commonly denotes the presence of organic disease of the stomach.

In chronic atrophic gastritis a total failure of secretion (achylia) may occur, and a similar abnormality is characteristic of established gastric carcinoma and pernicious anæmia.

No aberrations are more difficult of determination because, from time to time, they occur without any disturbance of the health. On the other hand, an unsatisfactory condition of health, with lowered resistance to various maladies, is frequently present. Anæmia, muscular debility, loss of appetite, flatulence, disordered action of the bowels—especially diarrhœa, bad complexion, clamminess and coldness of the hands and feet, vaso-motor disturbances, and many signs of auto-intoxication may be evident. These symptoms are so common and so vague that rarely are they assigned to their true cause. In all such obscure or indefinite cases an examination of the gastric chemistry should never be neglected, for it alone may reveal the nature of the condition and indicate the appropriate treatment.

It is certain that many of these cases of hypochlorhydria are functional neuroses and not primarily diseases of the stomach. That nervous disturbances such as fear, violent emotion and shock can produce temporary arrest of gastric secretion is demonstrable. I have seen numerous cases where merely the anxiety evoked by the anticipation of the extraction of the test breakfast suffices to produce a temporary achylia. That this has been the sole cause has been proved by finding a normal secretion when these patients were kept in ignorance that the breakfast was a test one.

It has been suggested that subacidity may be due to defect of the hormone or hormones which stimulate gastric secretion.

There are undoubtedly a large number of allied conditions which are characterised by diminution of the gastric secretion. By various terms an attempt has been made to differentiate these. From the therapeutic and prognostic point of view it is of some importance to appreciate the nature of the alteration in the gastric secretion.

It will be obvious that the diminution in secretion may be confined to the HCl, to the enzyme, or to both HCl and the enzyme. If HCl alone is depressed or absent, the condition should be called hypoacidity (subacidity) or achlorhydria (total suppression). If the ferment alone, apepsia, and when there is a loss of both HCl and pepsinogen, achylia. It will be found in practice that these conditions are often only stages in the evolution of the disorder and that from a diminution we gradually proceed to a total extinction of gastric secretion.

Clinically it should be noted that when there is total absence of HCl we may usefully call the case one of achylia gastrica. For even though the acid alone be wanting, and the ferment present, the latter remains inert from want of its activator.

Achylia Gastrica : Simple or Benign Achylia.—This term has been rather loosely employed by various writers to denote several conditions, the main feature of which is the apparent absence of hydrochloric acid from the gastric secretion. But in its truest sense it should really mean the absence of free hydrochloric acid and the ferments. It would be better if the term Achlorhydria were universally employed for those conditions in which only the absence of free acid had been determined, and Achylia retained for all associated with the absence of acid and the ferments. It will at once be seen that the distinction between the two conditions has

considerable importance clinically from the prognostic and therapeutic points of view. The term Achylia is, however, a convenient one for those cases in which free hydrochloric acid is absent from the gastric secretion, and, although not scientifically accurate, it may be usefully retained till the pathogenesis of the disorder has been more definitely defined.

We owe the term Achylia to Einhorn, who employed it to denote the absence or gross diminution of free hydrochloric acid in the gastric secretion. It will usually be found that in most cases the ferments may not be wholly suppressed. It is right, in certain cases where doubt as to the true nature of the condition exists, to examine the gastric filtrate for the presence of rennin-zymogen and pepsinogen. It should be noted that these substances are not necessarily absent because no indication of their presence is obtained from the macroscopic characters of the extracted test meals. Obviously their digestive powers remain latent in the absence of their activator —the hydrochloric acid.

I am convinced that achylia is not sufficiently recognised as a causative factor in many apparently obscure gastro-intestinal derangements. In investigating the gastric chemistry of hundreds of cases to elucidate the cause of certain derangements of the digestive tract, I have been rather struck by the relatively large number in which achylia has been revealed. This at once raises the consideration of the conditions under which this disorder may be found.

The etiological factors are not by any means uniform.

The term *Achylia Gastrica Nervosa* attempts to express the cause of one common variety in which, apparently, the condition may be regarded as a neurosis of secretion. Probably this is the simplest and most favourable form of the disorder, and, as might be expected, it occurs more frequently in women than in men. It is not, however, necessarily associated with other signs of neurosis or neurasthenia. I have frequently found it in the young and apparently perfectly healthy subject. The nervous origin of many cases is, however, apparently well established. That so great a failure in the gastric secretion may be thus brought about is proof of the important rôle played by psychic and emotional disturbances in gastric disorders. I am certain, from frequent observations, that the condition of achylia is often quite temporary and evanescent. For example the emotional disturbance produced in nervous people from the

dread of the " stomach pump " is capable of temporarily suppressing the gastric secretion. In this way wrong conclusions may be drawn from the analysis of the extracted meal. The advantage of giving the test meal without the patient knowing for what purpose it has been administered will thus be obvious. Ample experimental corroboration of these observations is forthcoming in the interesting work of Bennett and Venables published in the *British Medical Journal* of October 30th, 1920.

I have recently seen a number of cases of this type of achylia arising from the grief of bereavement. It is also well known that excessive fatigue may temporarily suppress the secretion of gastric juice.

On the other hand, as has been pointed out by Edkins, a hormone elaborated in the gastric wall acts as an excitant of gastric secretion quite independently of the action of the nervous system. Under what conditions this substance is elaborated and controlled we do not know.

Besides, achylia would appear to be often associated with various conditions in which a neurosis appears to play no part. I have noted that it may be found in many chronic and wasting diseases. In chronic tuberculous affections with amyloid change, cirrhosis of the liver, chronic parenchymatous nephritis, profound anæmias, mucous colitis, and especially in diabetes mellitus, to mention only a few, achylia is not infrequently present.

In pernicious anæmia we find the largest incidence of simple achylia. All my hospital cases of pernicious anæmia are examined for this abnormality, and I have never yet noted its absence in this blood disease. Probably it is a very early symptom, and in all cases of primary anæmia in adults the detection of achylia may be regarded as an important diagnostic feature of this grave malady. That the achylia which may be associated with it has no pathogenic influence in the causation of pernicious anæmia may be conclusively inferred from the fact that hydrochloric acid may be absent from the gastric juice for years without anæmia or any other disturbance of health.

Achylia is also commonly the result of well-defined organic changes in the mucosa due to chronic gastric diseases. In chronic catarrhal gastritis, from alcoholic excess or other causes, achylia gradually comes on. In all conditions associated with atrophic changes in the mucous membrane, such as the so-called " phthisis

ventriculi" or in "sclerosis ventriculi," the secreting glands are slowly destroyed and a permanent achylia is produced.

The achylia of cancer (malignant achylia) stands in a position by itself. Its features are characteristic and diagnostic. (*See* Cancer of Stomach.)

Before a diagnosis of achylia is made, the gastric contents should be repeatedly examined.

The differential diagnosis of simple from malignant achylia must always be attempted. The main diagnostic points of the latter are as follows :—

Pain, constitutional disturbance (loss of weight and cachexia), blood in gastric contents or fæces, and tumour formation. These symptoms are usually absent in benign achylia.

Diagnosis.—There is no known clinical picture of this condition. Sometimes it is discovered accidentally, and at other times as the result of an investigation. Its presence can only be affirmed after chemical examination of the gastric filtrate. When a test breakfast is given for this purpose and extracted in one hour the macroscopic appearances of the returned meal are usually quite informing. The toast of the Ewald's breakfast has undergone no digestion and no solution. Each particle is discrete, and the toast has only the appearance of being macerated. It is not converted, as it should normally be, into a homogeneous liquid resembling peasemeal gruel. In achylia the constituents of the meal are too readily recognised, and there is practically no digestive change.

The impossibility of properly withdrawing a test meal without the aid of an extractor will be well demonstrated in most of these cases. The return is very small in most cases of achylia simplex, is thick, and is withdrawn from the stomach with difficulty. This form is sometimes spoken of as dry achylia. Indeed, at the end of one hour the meal may have entirely left the viscus. There is no condition in which the stomach may more rapidly empty. These features are due to (1) the patency of the pylorus resulting from the absence of hydrochloric acid. (2) to some extent a more active peristalsis, and (3) to the failure of the gastric secretion to add to the bulk and the fluidity of the meal.

In the simplest type of achylia—achylia nervosa—the returned meal is free from mucus, has no offensive odour, and is usually neutral in reaction to litmus paper. But from time to time the returned meal may be normal, or even excessive in quantity,

particularly in those cases associated with gastroptosis and other disorders of motility. In such cases, and also in atrophic gastritis, mucus is often abundantly mixed with the ill-digested test meal, making the whole a gelatinous mass.

The filtrate should be tested with (1) blue litmus paper: if an acid reaction be present this point should be noted. (2) Congo red paper: in the absence of free hydrochloric acid there will be no blue tint on the congo red paper. If the blue litmus gives a strong acid reaction and the congo red no blue colouration, organic acid is present. The total acidity should also be estimated. It will probably be found to be very low in simple achylia, and in this respect in striking contrast to the relatively high total acidity of malignant achylia.

Having determined the absence of free hydrochloric acid a further investigation becomes necessary. In the graver and permanent achylias due to atrophic changes in the mucosa there may be total destruction of the secreting glands. In such cases no hydrochloric acid at all can be secreted. To determine then that none has been secreted, when none is present free, the combined acid must be sought for. The Sgœqvist or other test* must be employed for this purpose. The importance of this investigation is twofold. The presence or absence of combined acid enables us to formulate a correct prognosis. If combined hydrochloric acid be present, clearly some acid is being secreted, and the glands are not wholly inactive or completely destroyed. Under treatment such cases may be expected to improve or recover.

Achylias thus resolve themselves into total or partial—the former, associated with the total suppression of the secretion of hydrochloric acid, usually permanent and incurable; the latter, with the presence of combined acid, showing some retention of the secreting function of the hydrochloric acid glands, may be regarded as hopeful under appropriate treatment.

Symptomatology.—The symptomatology of achylia gastrica is very variable. At times it is associated with no aberration from the normal health. Generally speaking, however, the clinical picture may present symptoms of (1) gastric disorder, (2) intestinal disturbance, or (3) gastro-intestinal derangement.

It may at once be said that gastric symptoms are uncommon alone, but that intestinal disturbance of one kind or another

* See tests for Combined HCl, pp. 122-123.

prevails. Perhaps the principal reasons for derangements of intestinal digestion are the rapidity with which the non-prepared meal is thrown into the bowel, and the absence of the normal acidity of the chyme. Sometimes after gastro-enterostomy, in cases where the stomach empties with precipitate haste, very similar intestinal symptoms to those found in achylia may, for a time, be present. Thus both the mechanical and chemical abnormality tend to produce disorder in the bowel. The badly dissolved meal and the altered chemical reactions are sufficient to account for the abnormal conditions produced in the bowel. One has only to recall the potent influence which hydrochloric acid has on the secretions of the duodenum, the bile, and the pancreas to realise the widespread disturbance which may follow its suppression. Its other and not less valuable influence as an antiseptic to the gastro-intestinal tract must always be borne in mind.

Those symptoms which are gastric are very vague and do not form clinically a recognisable symptom-complex. But I have dealt with a number of cases in which there was gastric pain coming on shortly after food, with considerable flatulent distension with belching of wind, bad or capricious appetite, thirst, and, not infrequently, nausea and vomiting.

Paradoxical as it may seem, in a trouble originating in the stomach, the principal and most common symptoms are due to intestinal disorder. In their order of importance diarrhœa stands first. The type is that of the diarrhœa of ingestion. Thus it frequently follows a meal and gives rise to the belief on the part of the patient that his "food is running through him." But not at all uncommonly the diarrhœa appears to have no relationship to the act of eating. It may have all degrees of severity. At times it is rather intractable. The stools are sometimes offensive, and in their bulkiness are out of proportion to the food taken. Macroscopically or microscopically they may be seen to contain remnants of ill-digested food such as meat fibres. Contrary to what one might expect, the diarrhœa of achylia is not so frequently associated with loss of flesh as that from many other causes. Indeed, the general nutrition may remain good and no signs of anæmia be present.

In long-standing cases of achylia with intestinal disorder mucous colitis may supervene. On the other hand, it should be noted that achylia gastrica is not uncommonly an accompaniment of mucous colitis.

Constipation, although less common than diarrhœa, is not an infrequent symptom. It is more difficult of explanation, but probably arises from a failure of proper motor innervation. As might be expected, diarrhœa and constipation may alternate.

Flatulence, both gastric and intestinal, may be a prominent complaint. All the usual subjective symptoms of gaseous distension, such as vertigo, palpitation, and colic may be present.

The urine in most cases of simple achylia shows no special variations from the normal. It may contain traces of acetone and show an excess of indican, but these substances are not found so commonly nor in anything like the same amount as they are in malignant achylia.

In middle-aged and elderly people achylia should be regarded when occurring for the first time, and even when it has none of the characters of the malignant type, with some suspicion. I have now had a considerable number of such cases under observation, and in a number of these malignant disease has been definitely determined.

Treatment of Achylia.—It will be understood that the exact treatment which is to be adopted will depend on the possible cause of the condition. In all cases which can be classified as achylia gastrica nervosa, and all those in which the achylia is partial, a good result may be expected. Its application should be directed to (1) the improvement of the general condition, and (2) to the restoration of the normal gastric secretion.

1. *General.*—Any obvious adverse hygienic condition should be removed. Fresh air, gentle exercise, avoidance of fatigue and over-excitement are essential to success. If there be loss of weight, rest in bed and general massage with olive oil have yielded me excellent results. This is especially the case in those subjects who are distinctly neurasthenic. For these a complete and rigid course of Weir-Mitchell treatment may be followed by rapid progress towards recovery. After the massage physical culture, in the patient's own home, should be carried on persistently. Müller's exercises are quite suitable for most cases, and their systematic use improves the nutrition and the gastro-intestinal functions. I have not been able, so far, to convince myself that high frequency or other electrical treatments have had any curative influence.

The diet must be properly selected. The exact ingredients chosen will depend on the nature of the gastro-intestinal symptoms which may be present, especially on the presence or absence of diarrhœa. A good diet should not only be a nutritious form of alimentation, but it should aim at having a therapeutic influence.

The most thorough mastication should be insisted on so as to get the maximum amount of insalivation and the greatest amount of starch digestion in the stomach. Starches are usually very well borne in simple achylia if their mastication be complete. To ensure this end they should be eaten dry. Other articles of diet and of good nutritive value which stimulate the gastric secretion must have their place in the menu. Extractives, soups, raw meat juice, raw eggs, plenty of salt and spiced foods all tend to increase the gastric flow. Depressants of secretion, such as large quantities of fat and oils, are not indicated in their crude state. I have found, however, that small doses of cod oil emulsion or of malt and oil are, in suitable cases, well borne and improve the nutrition and general tone.

A suitable dietary for cases without diarrhœa, and in which catarrhal gastritis is absent, would be as follows :—

At 8 a.m.—*Breakfast.*—Two eggs or white fish, etc., rusk or toast, small cup of tea or *cafe au lait.* Dose of artificial digestive.

At 11 a.m.—Dry biscuit, cracknel, rusk, toast, etc., chewed till absolutely liquefied in the mouth. When finished a glass of water, milk, or bouillon to be sipped very slowly.

At 1 p.m.—*Dinner.*—Strong soup (ox-tail, beef tea, etc.). Meat underdone and grilled. Stewed fruit. Biscuit and cheese. Dose of artificial digestive.

At 4 p.m.—Another farinaceous meal (dry potato, toast, stale bread). When finished, fluid as may be prescribed.

At 6 p.m.—*Supper.*—White fish, cheesesoufflé, omelette, small portion of stale bread. Dose of artificial digestive.

At 10 p.m.—Bowlful of gruel, Benger's food, or some other starchy material to be sipped slowly.

If any signs of gastric fermentation be present gentle lavage, twice weekly, with normal saline solution at 100° F., is very useful. This is specially so in catarrhal affections, with achylia, associated with the accumulations of tenacious mucus.

Hydrotherapeutic measures are also to be recommended. The employment of hot compresses, Aix-massage-douche, and the alternate use of cold and hot douches over the gastric region are all useful therapeutic measures.

2. *Medicinal treatment* aims at stimulating the gastric glands to a better secretion. Bitters are sometimes useful, but alone are not sufficient. By far the most important therapeutic agent we possess is hydrochloric acid, more especially when it is combined with pepsin. If it be kept in mind that about three pints of gastric juice are normally secreted in twenty-four hours, and that hydrochloric acid is present in from 0.1 per cent. to 0.2 per cent., it will be seen that the dose of dilute acid must be very large. Indeed, it is not practicable to administer all the acid represented in the normal output. In cases of permanent total achylia the administration of hydrochloric acid must be as continuous as is the administration of thyroid in myxœdema. But in all those in which the glands are not destroyed, but only functionally inactive, the patient's gastric contents should be tested at intervals to avoid the possibility of producing an artificial hyperchlorhydria. The dilute hydrochloric acid should be administered in one dose after the three principal meals. These should contain all the proteid material which is to be administered in the day's dietary. These three meals should be taken at five hourly intervals. Starchy material should not form a large proportion of their contents. The carbohydrates should be partaken of in the intervals—three hours after each meal—and should, as far as possible, be eaten quite dry, and any fluid which may be desired taken when the solids are finished.

It will be found in practice that perhaps the best form for administration of the artificial digestive is :—

℞ Acidi hydrochlorici diluti .... ℥i-ii
Glycerini pepsini .... ℥iiss.
Extracti condurango liquidi .... ℥i
Aquæ chloroformi .... ad. ℥vi.
M.

Signa :—℥ii, in water, thrice daily after food.

If any fermentation is evident, a small dose of resorcin may be added to the above. Various other bitter tonics may also be tried from time to time during the treatment. Sometimes strychnine is useful, with or without physostigmine. In cases where there is

motor insufficiency, as in the achylias with gastroptosis, this combination will be found useful :—

℞ Strychninæ .... gr. $\frac{1}{24}$
Physostigminæ Salicylatis .... gr. $\frac{1}{96}$
Quininæ Hydrochloridi .... gr. i
Extracti Euonymi Sicci .... gr. $\frac{1}{4}$
Gingerine .... gr. $\frac{1}{4}$

Ft. pil. mitte tales, 60. M.

Signa :—One pill thrice daily after food.

In the treatment of pernicious anæmia we are too often content to administer arsenic alone, and to think that no further medical treatment is of any value. But much good can be done to prevent toxæmia and improve the blood in proportion to our ability to remove the accompanying achylia or to compensate for its presence. Many of the severe gastro-intestinal symptoms which so rapidly deteriorate the patient are due to the accompanying achylia. In all cases of pernicious anæmia the treatment for achylia, in its fullest extent, should never be omitted. To the artificial gastric juice may be added an appropriate dose of the acid solution of arsenic.

Heterochylia signifies a condition, not too well identified, in which there is irregularity in the gastric secretion, subacidity and hyperacidity alternating with each other. Such aberrations, I believe, occur alone in neuropathic subjects and are, in this respect, akin to the achylia of youth. As a rule no symptoms of a diagnostic kind are present. An examination of the gastric contents, carried out from time to time, is necessary to disclose these alternations.

The prognosis is usually good, and treatment consists in hygienic measures, suitable diet, and tonic remedies.

Diminution or Excess of Mucous Secretion.—Diminution of mucous secretion is probably only a concomitant of other disorders, and does not exist by itself. An absence or serious diminution of mucus is commonly spoken of as **amyxorrhœa gastrica.**

A too scanty secretion of mucus deprives the gastric mucosa of its normal protection against irritants. This condition probably plays a rôle in the etiology of peptic ulcers and determines to some extent their site. Amyxorrhœa is commonly a feature of peptic ulcer and it is found in hyperchlorhydria and gastro-succorrhœa and

predisposes to the auto-digestion of the mucosa by an over-acid and active gastric juice.

This condition is best treated by lavage with 1 in 1,000 or 1 in 2,000 solution of silver nitrate, and by the administration of a little salt solution before each meal. If the amyxorrhœa be only an accompaniment of hyperacidity, the latter condition should be dealt with. As a rule it will be found that the secretion of mucus is in inverse ratio to the secretion of HCl.

Myxorrhœa Gastrica or Gastro-myxorrhœa (excessive secretion of mucus) is, of course, a usual feature of the various forms of chronic gastritis or catarrh of the stomach. Large quantities of mucus are also commonly found in the vomitus and in the returned test meal in carcinoma of the stomach.

The best method of determining increase in the amount of mucus is by the test meal. Discrete particles of mucus are usually contaminations from the naso-pharynx or the pulmonary tract; but when mucus is abundant and intimately mixed with the vomitus or test meal it may be regarded as coming from the stomach and indicative of catarrh. Proof of its gastric origin is afforded by the detection microscopically, in the mucus, of numerous columnar epithelial cells. An analysis of the test meal in such cases will also likely show a diminished or absent HCl content except in the 'acid gastritis' of Boas.

Transient catarrhs of the stomach with pronounced symptoms, headache, prostration, and uncontrollable vomiting of glairy mucus, are comparatively unimportant. Such attacks expend themselves in a short time.

Permanent gastro-myxorrhœa is a typical feature of chronic (catarrhal) gastritis. Its significance, prognosis, and treatment have been already considered in the chapter on chronic gastritis.

CHAPTER XXII.

DISORDERS OF MOTOR FUNCTION.

Gastric peristalsis, or the rhythmical contractions which are associated with the expulsion of food from the stomach, may be diminished or increased.

Normally the stomach has a certain degree of elasticity and tonus which resist over-distension. Thus in health the meal is embraced closely by the gastric wall and any undue accumulation of gas is expelled. But the muscular power and elasticity may become impaired and all degrees of atony result with signs and symptoms commensurate to the amount of change.

When pyloric stenosis begins to form, and as obstruction progresses, compensatory hypertrophy of the gastric muscle results. As long as compensation remains intact we shall have over-powerful and excessive contractions, usually taking the form of visible peristalsis accompanied by pain.

Later on, when compensation begins to fail, the peristalsis diminishes, and atony supervenes.

Apart from such alterations in the peristalsis, disorder of motor function includes spasmodic affections localised to the orifices (cardio- and pyloric spasm) or involving the greater part of the stomach (gastrospasm).

1. Myasthenia Gastrica.

Myasthenia gastrica has been variously designated *motor insufficiency, myasthenia ventriculi, motor insufficiency of the first degree* and *gastric atony*. All these terms are employed to indicate loss of muscular power.

This condition may be primary or secondary to pyloric stenosis.

Of the dynamic affections of the stomach, myasthenia or atony is of very frequent occurrence. It is one of the gastric disorders in which heredity plays an important part, and, like duodenal ulcer, it is often familial. It may be a legacy from infancy.

Gastric atony may be acute or chronic. The chronic condition, although it may primarily be of nervous origin, is associated with muscular weakness from degeneration.

Gastric atony is most commonly met with in the neurotic subject, and is seen more frequently in the sedentary and urban worker than in those leading an out-of-door rural life.

All kinds of mental disturbances predispose to this affection. Thus, violent emotion, grief, anger, and over-work may evoke the condition. Even in those who have no special tendency to the disease a temporary myasthenia may be thus produced, and it may follow various acute infections—especially diphtheria and enteric fever. The dyspepsia of phthisis is frequently due to this condition, and it may be found as an accompaniment of any wasting disease.

Or myasthenia may be acquired by prolonged overtaxing of the stomach from excesses in food or from the bad habit of drinking excessive volumes of fluid with meals. Under such conditions the musculature gives way and atony follows.

More severe types of myasthenia gastrica are those secondary to carcinoma, duodenal ulcer and chronic gastritis.

The importance of the affection turns on the presence or absence of stagnation and the degree to which it exists. In myasthenia gastrica there is, as a rule, a retardation in emptying beyond the normal time. In severe cases the stomach may only get rid of its contents before the next meal is due, or in still more advanced conditions it may never empty completely during the night.

In simple gastric atony there need be no dilatation of the stomach, although the two conditions are not infrequently associated. Gastric atony may be the forerunner of dilatation. The two conditions are often confused, perhaps because they are so closely related.

The motor functions of the stomach may most readily be investigated in these three ways :—

(1) By test meals.
(2) By certain chemical tests.
(3) By X-ray investigation.

Acute Gastric Atony.—This affection may be complicated by acute gastric dilatation, or exist by itself. It is not a very common condition, and arises chiefly as a sequel to indiscretion in eating. Bolting a too large amount of food with an excessive amount of fluid may be the determining factor. The musculature gives way,

resulting in gastric distension, undue retention of the meal, with marked succussion and some tenderness over the gastric region.

The Symptoms are somewhat variable, but pain, sense of fulness, and belching of wind are among the commonest. The discomfort may persist till vomiting occurs. It is of the ' delayed ' type.

The Treatment consists in abstention from food, evacuation of the stomach contents by the tube and subsequent lavage. The administration of a brisk purgative—*e.g.*, five grains of calomel—will be found beneficial. Prophylactic treatment consists in restricting the amount of food. The solids should be eaten first, very slowly, and any fluid permitted ought to be taken at the end of the meal. In this way it is difficult for the large eater to steal a march on the stomach.

Acute gastric atony is not in itself a serious condition, but as it may develop into the more dangerous acute gastric dilatation it ought to be treated without delay.

Chronic Gastric Atony.—As already explained, the urgency of this condition is directly proportionate to the amount of stagnation and retention which may accompany it.

If the stomach empties within a reasonable time—*i.e.*, between meals—a small amount of myasthenia is unassociated with symptoms. Such cases are from time to time the victims of an attack of acute atony with symptoms as described above. Chronic cases owe their importance to the degree of stagnation present.

Symptoms are variable, and the clinical picture is progressive. Fulness after food—both solid and fluid alike—a feeling of satiety long before a meal is completed, belching of wind and eructation of mouthfuls of fluid are among the early and common symptoms of a mild degree of retention.

On *examination of the gastric region* a variable amount of tympanitic distension of the stomach, with succussion, may be detected. If the splashing is very extensive in area and is detected well below the umbilical region, it may be construed as indicative of an associated dilatation. The test breakfast in these cases will be found to be retained too long, and a very large return may be extracted, often over 100 c.c.

If such a case be left untreated it tends to progress, and the symptoms and signs undergo aggravation.

When stagnation is prolonged, the features of hyperchlorhydria and auto-intoxication may be added. Pain and vomiting may then become prominent, the appetite fails, constipation becomes obstinate and various nervous and circulatory disturbances may develop. Except where there is associated organic disease little loss of flesh occurs.

In such severe cases the physical signs are pronounced. Splashing can be elicited for hours after even a fluid meal, and the test meal will have certain characteristic features of retention. The longer the meal remains in the stomach the more gastric secretion it will evoke. Thus we get a very large return due to secretion being added to the test meal which the stomach fails to pass into the intestine.

The usual test supper of currants taken late at night should be followed by Ewald's breakfast early next morning. The breakfast should be extracted one hour after its ingestion, and the amount returned noted. By its general appearance, the presence or absence of currants, the chemical analysis and the microscopic examination data on which a diagnosis may be based will usually be available.

If the return is excessive and contains currants, we have either severe primary atony with stagnation or obstruction at the outlet. When the contents are set aside in a cylinder there will not be a separation into three layers if we are dealing with atony. In obstruction at the outlet this separation is usual.

On chemical examination of the test meal from such a case an abnormally high percentage of free HCl may be found, or the total acidity may be very high from an undue amount of combined HCl. The presence of an abundant hydrochloric acid secretion prevents the development of an abnormal flora and, as a rule, few organisms will be found. Thus there is a striking difference between the stagnation of myasthenia and the retention due to obstruction at the outlet. In the latter there are numerous sarcinæ and perhaps yeast cells if the stenosis be simple, while if it be malignant, Oppler-Boas bacilli and lactic acid will commonly be found.

When HCl is habitually low or absent, the suspicion that carcinoma may be present ought to be entertained.

In adynamic conditions of the stomach, with an absence of HCl, the pylorus is in a state of relaxation, and the stomach may empty itself with abnormal rapidity. These cases should not deceive the physician and lead to a wrong diagnosis. (*See* Achylia Gastrica.)

Treatment.—The prophylactic treatment of chronic gastric atony is by far the most important. The aim should be to prevent the early stage of this disease advancing till symptoms are chronic and severe.

If it be established that myasthenia is due to cancer, tuberculosis, or chronic gastritis, the treatment should be directed to alleviate these conditions.

Of the idiopathic cases the most intractable are those in which there is a pronounced familial tendency.

The best preventive treatment is to establish a good mode of living and to advise an out-of-door life. Horse riding, physical culture (Müller's exercises), electrical treatment, hydrotherapy, careful dietary, thorough mastication, strict moderation in the use of alcohol and tobacco, are among the best preventive measures.

Should it be found, from examination of the stomach contents, that any important degree of stagnation be present, the stomach should be washed out at bedtime and a light supper given shortly after. For the lavage various antiseptics in solution can be employed, followed by sterile water. The lavage may be continued nightly for two weeks.

Except in intractable cases I am wholly against the use of support by mechanical appliances. Abdominal belts and supports only tend to weaken the muscles. Carefully thought out exercises and massage to promote the development of the abdominal muscles are ultimately of far greater value.

I have seen great benefit result from making the patient lie on the right side during sleep, with the lower end of the bed raised so that the stomach and intestines are prevented from drooping. When this procedure is carried out for a lengthened period of time it has a marked influence in facilitating the emptying of the stomach and restoring its tonicity, as it has in cases of gastroptosis (*q.v.*).

I have tried in numerous cases the effect of frequent small meals at short intervals and of full light meals at longer intervals. These have been both dry and fluid. My experience of these makes me have no hesitation in recommending three moderate meals, to be eaten quite dry, with a restricted amount of fluid three to four hours after ingestion.

As all these cases are associated with some delay in gastric evacuation the introduction of small, even though fluid, meals keeps up a continuous functional activity and so promotes

exhaustion. To administer food at rather long intervals and give the stomach the chance to empty between the meals is the better plan. I have found the following diet, taken at fixed intervals, quite satisfactory :—

8 a.m.—*Breakfast.*—One soft boiled or poached egg ; one to two slices of toast with fresh butter ; all to be chewed thoroughly.

12 noon.—A glass of hot or cold water to be sipped slowly, or, if the state of nutrition be unsatisfactory, a cup of café au lait, chocolate made with an equal part of milk and water, or a glass of Horlick's milk may be advised. Not more than eight ounces of fluid should be taken.

2 p.m.—*Lunch or Dinner.*—White fish, chicken, tripe, underdone tender chop, or fillet steak, sweetbreads and such like may be chosen for dinner. Well-boiled macaroni is preferable to potatoes. Custard pudding and a roasted apple may follow the meat.

6 p.m.—Eight ounces of fluid may be again taken.

8 p.m.—*Dinner or Supper.*—Similar to lunch at 2 p.m.

11 to 12 (midnight), or any time during the night if the patient be awake, eight ounces of fluid may be taken.

In most instances I have found such a dietary to agree well. If the patient's nutrition be very poor it may be necessary to augment its nutritive value by the addition of some foods to the fluids. Many of the preparations on the market, such as Ovaltine, Sanatogen, etc., may be used for this purpose.

Milk will not always be found to be the lightest food. When marked hyperchlorhydria is present, hard and indigestible clots are formed, which may not quickly leave the stomach. In all such cases the milk should be treated. The addition of Ovaltine, Mellin's food, lime water, sodium citrate or barley water, have the power of preventing massive coagulation. Liquid extract of malt, which is also nutritive, may be added to effect a similar purpose. Egg flip, made with only a small amount of milk, will be found specially beneficial for cases in which superacidity is marked. Virol is usually well tolerated, and may be taken with the fluids. Olive oil before food is useful as a nutrient, aperient, and antacid.

The urine should be watched, and if any tendency to over-concentration and precipitation be seen, a larger amount of fluid may be advisable. To avoid gastric distension it may be found necessary to administer it rectally, and a little glucose may be added. If this method is adopted, it is best given when the patient retires to bed at night.

If the symptoms are urgent, the patient, certainly at the outset of treatment, should be confined to bed. If absolute rest is not considered essential, the assumption of the recumbent position for half an hour after each meal, and when possible after each drink of fluid, should be prescribed. While the erect position is maintained the fluid tends to sag the stomach downwards, distend the duodenum and delay emptying.

After each fluid meal the patient should be instructed to lie down on his right side.

In simple myasthenia gastrica lavage is not usually indicated, but cleansing the stomach once a day with a pint of warm water is helpful if stagnation be pronounced.

Certain drugs co-operate in promoting a better muscular power of the stomach. These should be given in small doses short of producing physiological action. Over-excitation of a weakened muscle must only increase its exhaustion. Strychnine, Physostigmine, and Ergot are all useful excitants of unstriped muscle. These may be combined with general tonics. If hyperchlorhydria be a feature of the case, atropine may be usefully used. The following formulæ I have found suitable :—

℞ Extracti Euonymi Sicci .... gr. $\frac{1}{4}$
Ferri Arsenatis .... gr. $\frac{1}{36}$
Strychninæ .... gr. $\frac{1}{36}$
Extracti Physostigmatis .... gr. $\frac{1}{4}$
Extracti Taraxaci .... gr. iss.
M.

Ft. Pil. Mitte Tales 60.

Signa :—One pill, thrice daily, after food.

℞ Sodii Arsenatis .... gr. $\frac{1}{36}$
Ferri Sulphatis Exsicc. .... gr. $\frac{1}{4}$
Extracti Belladonnæ .... gr. $\frac{1}{4}$
Extracti Rhei .... gr. i
M.

Ft. Pil. Mitte Tales 60.

Signa :—One pill, thrice daily, after food.

The aperient drugs in these pills may be withdrawn or increased according to the requirements of the patient.

Should it be demonstrated that hypochlorhydria be a feature of the case, hydrochloric acid is indicated.

2. Acute Gastric Dilatation.

Acute gastrectasis may be a sequel of acute atony. It has occasionally been noted to follow excessive meals, injuries to vagus, diminishing motor innervation (as in head injuries), obstructive interference with the pylorus (pyloro-spasm and stenosis), various toxæmias due to septic and infectious diseases causing gastric paralysis and as a post-operative condition.

Of all these causes the only one which is of supreme importance is the last—*post-operative dilatation.* It is of fairly common occurrence, and the resulting condition is extremely dangerous, and often rapidly fatal. While most common after laparotomy, acute dilatation may occur after operation on any part of the body.

In the acute tympanitis of enteric fever the stomach may participate in the general paresis of the gastro-intestinal tract, and in the ' phantom tumour ' of hysteria there is a general dilatation of the whole digestive tube from altered innervation.

The exact causation of acute dilatation of the stomach is still a controversial subject, and many theories have been advanced to explain its occurrence. Of these explanations the more important are :—

(*a*) *Nerve Lesions produced either by Trauma or Toxæmia.*—Injuries to the brain and cord undoubtedly may cause gastric paralysis, and it may be an accompaniment of ' shock.' We have many examples of paresis following, notably, diphtheria, enteric fever, and other infectious diseases, and possibly chloroform toxæmia, or the auto-intoxication of gastric fermentation. Toxic agents may thus act on the nervous apparatus or on the musculature of the stomach. The condition has also been noted in cases of tetany, chorea, epilepsy, and hemicrania.

(*b*) *Mechanical interference with the pylorus or duodenum.*—Pylorospasm, resulting from hyperchlorhydria, has been described as a causative factor in the production of acute gastric dilatation.

After abdominal operations when, from the use of preparative enemata or purgatives, or from starvation, the intestines are empty, they may fall into the true pelvis and thus cause traction to be exerted on the root of the mesentery and the superior mesenteric vessels, producing a mechanical obstruction of the duodenum. Albrecht, who advanced this explanation, considered that the fall of the intestine *alone* was insufficient to produce the amount of obstruction necessary to cause acute gastric dilatation, but that it

caused sufficient obstruction to result in over-distension of the stomach. In my opinion there must be a primary weakening of the gastric tonus and elasticity to permit of slight mechanical interference becoming operative. In support of this view there is evidence that the gastric distension is primary and that the intestine is pushed downwards into the pelvis from pressure from above. In such cases a paresis of innervation or of the gastric muscle directly, from toxæmia or trauma, is doubtless the primary pathogenic factor, while a secondary one may be the arterio-mesenteric constriction due to the traction of the depressed intestine. These views are supported by von Herff and others.

Again, over-distension of the stomach beyond its power of control may cause secondary pressure on the duodenum and precipitate acute gastric dilatation. I have myself, on two occasions, produced acute gastric dilatation while performing diagnostic inflation with sodium bicarbonate and tartaric acid. In both the cases there was an antecedent myasthenia gastrica. Various other views have been advanced, but they are not of sufficient importance to require special notice.

Clinical Picture.—The milder degrees of acute gastric dilatation may be associated with no urgent symptoms, but when the condition is pronounced they are severe.

Young adults who have undergone operation are most apt to be attacked.

The onset is sudden, and the attack may come on some days or weeks after operation, when there is nothing to suggest that the patient is not making good progress towards recovery.

Sometimes symptoms are latent, but as a rule there is sudden abdominal distress, with a feeling of distension and pain in the gastric region, persistent vomiting, faintness and collapse.

The facial expression is one of anxiety. The skin is cold and clammy, and the general appearances are not dissimilar to those seen in angina pectoris. The pulse becomes small and rapid, the respirations shallow and accelerated. The temperature may be subnormal and the urine scanty.

Physical examination of the abdomen will usually reveal distension, mostly on the left side. Sometimes the entire outline of the stomach can be seen through the abdominal wall.

Succussion may be elicited if fluid as well as gas be present in the stomach. Visible peristalsis is not commonly seen, except in

acute dilatation from pyloric stenosis. Indeed, active peristalsis, indicative of good muscular action, is rather incompatible with acute dilatation. A very tympanitic note will be elicited by percussion over the area of distension.

In acute gastric dilatation there is commonly a perversion of the chemistry. The gastric secretion may be suppressed and free HCl be absent. When duodenal or intestinal elements are returned through the stomach tube, the condition should always be regarded with much apprehension. The more nearly the gastric secretion approaches the normal the better is the prognosis.

In two cases which I have recently seen there was a marked acetonæmia.

If the introduction of the stomach tube and lavage remove the gas and the fluid which may be in the stomach, and distension forthwith diminishes, we have evidence of a gastro-duodenal type of dilatation. If the dilatation be largely of the gastro-intestinal type only partial relief will follow gastric lavage; while in those cases principally due to intestinal tympanitis the introduction of the stomach tube and gastric lavage will have little ameliorative action.

The prognosis depends on the early recognition of the condition and on the amount of response to treatment. In the most severe cases there is a fatal termination within a few hours. The death rate recorded amounts to about 70 per cent. With an early recognition and appropriate energetic treatment there should be a great reduction in this mortality.

Treatment.—Since we cannot predict which of our patients may suffer from acute gastric dilatation, certain steps might be taken to anticipate this complication. Fortunately, relatively to the large number of people who undergo surgical operations, this crisis is so comparatively rare that it may be considered unnecessary to adopt prophylactic treatment as a routine.

Nevertheless in all patients who have any signs or symptoms of myasthenia gastrica, or whose previous history is suggestive of this condition, certain measures ought to be initiated prior to operation. The stomach should be washed out on the two days preceding operation, and the patient should have the faradic current applied with one pole over the spine and the other, in the form of the wire brush, over the anterior gastric region. This will promote a better tonus of the abdominal wall. High frequency may also

be employed to stimulate contraction of the unstriped gastric muscle.

The bowels should be moved two days before operation with three to five grains of calomel, and certain medicaments given hypodermically. Of these I have found physostigmine sulphate (gr. 1/100) and strychnine sulphate (gr. 1/30), given morning and evening for a few days prior to operation, apparently prophylactic in their action.

The treatment of the actual attack must be energetic and drastic. The stomach should be immediately emptied by means of the stomach tube, and then washed out with water at 100° to 102° F. The process should be repeated at three hours interval till cessation of gastric redistension relieves anxiety.

Oral administration of food and drink must cease, and rectal injections of warm normal saline and glucose be relied on to relieve thirst and maintain the patient. If there be any tendency to collapse, some brandy (℥ii—℥iv) may be added to each injection, along with some liquid peptinoids.

Physostigmine and strychnine should be used as above, and to these may be added gr. 1/100 of atropine sulphate if supersecretion is taking place.

If collapse should be threatening, it may be necessary to give an intramuscular or intravenous saline. I have found, for impending syncope, 1 c.c. of pituitrin a most efficient remedy. It not only raises the blood pressure, but it exerts a most excellent tonic influence on the muscle of the stomach. It should be given hypodermically if the condition is urgent, or it may be added to the rectal enemata, as a preventive, in less severe cases.

Various mechanical measures may be adopted to mitigate upward pressure on the diaphragm and so prevent interference with cardiac and respiratory actions.

The upper end of the bed should be blocked up, and the head and shoulders well raised with pillows. It will be obvious that the exact position advised will depend on the type of the distension. To raise the patient over much may depress the intestines, and so, by increasing the pull on the duodenum, cause further mechanical obstruction of that part.

According to Kemp, the ventral posture is the best for the acute gastro-duodenal variety of dilatation. The patient should be kept lying on his belly over a soft pillow.

Operative treatment has been advocated for these conditions, but the depressing effects of the anæsthetic and the shock of the operation are certainly more likely to precipitate a fatal termination than to afford relief.

3. Chronic Gastric Dilatation.

By this designation should be meant a chronic dilatation of the stomach secondary to obstructive trouble at the outlet.

The terminology of this ailment is complex and confusing. It has been described by Boas as *motor insufficiency of the second degree*, and it has many other synonyms: *Ectasia ventriculi*, *chronic gastrectasis*, and *chronic dilatation of the stomach.*

Much care should be exercised to exclude error in the diagnosis of this condition. It is too commonly assumed that stomachs in which marked succussion can be easily elicited are dilated. Most stomachs, at certain periods after food, will splash on palpation, and yet there may be no real dilatation, but only minor atony.

Again, the ' dropped stomach,' gastroptosis, is not uncommonly erroneously diagnosed as dilated stomach. It is, however, true that a prolapsed stomach is also sometimes a dilated stomach.

In true dilatation only the lower border of the stomach descends, whereas in gastroptosis the upper border may be found depressed as far as the normal situation of the lower.

The mere size of the stomach and its capacity to hold large volumes of fluid are not the criteria of dilatation.

Megalogastria, or the ' large stomach,' may be produced as a result of frequent over-distension, from over-eating and drinking, or it may be a congenital affection. But the ' large stomach ' differs from the ' dilated stomach ' in the absence of symptoms.

The essential features of true chronic dilatation are increased capacity plus loss of function.

Idiopathic Atonic Dilatation of a chronic character has been described by some writers. It is a condition which must be of rare occurrence, apart from some interference at the outlet, and the retention which is associated with it is of a passing character. In certain cases it has, however, been demonstrated that the stomach is never completely empty from one meal to another.

The atonic type of dilatation is commonly associated with multiple nervous manifestations. Hysteria, neurasthenia, and insanity predispose to the condition. It will thus be seen that it has similar

etiological factors to those of chronic myasthenia gastrica (*q.v.*), which may be the precursor of chronic dilatation.

Treatment.—All the predisposing causes of myasthenia gastrica should, as far as possible, be removed. The strictest attention to general hygiene should be insisted on. Fresh air and adequate exercise must be prescribed. Dietary should be carefully regulated and all excess restricted. For the most part the meals should be small and concentrated, and fairly dry. Fluids should be taken between the solid meals. The dietary and regimen detailed for the treatment of myasthenia gastrica will be, for the most part, suitable (*see* page 305). Any modifications necessary to meet special conditions the physician will make for himself.

The examination of the gastric contents will determine the medicinal treatment. If hyperchlorhydria is prominent, appropriate measures will be required to deal with it (*see* Hyperchlorhydria, page 287). If achylia complicates the condition, it too will require special treatment (*see* Achylia, page 295).

4. Cardiospasm (Spasm of the Cardia).

This is one of the rather more important motor neuroses due to spasmodic muscular contraction of the cardiac orifice. It is usually associated with a varying degree of dysphagia.

Normally there is a certain amount of contraction at the cardia, which causes a trifling amount of delay in the passage of solids and liquids into the stomach. This resistance at the cardia is often experienced when the stomach tube is being introduced. Cardiospasm may be temporary or permanent. It may be a primary condition when it is purely a functional neurosis, or it may be due to reflex irritation of the vagus nerve or secondary to well-defined disease—*e.g.*, carcinoma.

Etiology.—The temporary form may arise from anything which causes local irritation. It may follow the attempt to swallow too large a bolus, too hot or cold fluids, or the passage of a bougie or stomach tube.

Cardiospasm is not an uncommon manifestation of hysteria and neurasthenic conditions. It is thus most common in women.

It has been also ascribed to atony of the muscular coats of the gullet, paresis of the vagus, primary œsophagitis and to a congenital predisposition.

Permanent cardiospasm is most frequently associated with local disease as peptic ulcer or cancer.

In all conditions where the spasm is lasting, dilatation of the œsophagus results. Some of the food is apt to accumulate above the constricted part and to be regurgitated at variable intervals.

Symptoms and Signs.—The primary neurotic type has a sudden onset, with sharp pain under the xiphoid. There is varying amount of dysphagia, and a corresponding amount of regurgitation. No gas is eructated during the attack. There may be some respiratory embarrassment.

Such attacks are at first of short duration, but in time they become prolonged and chronic.

If there be any doubt as to the nature of the symptoms, the passage of a bougie will yield reliable information as to the condition of the cardia, and an X-ray investigation will show the nature of the obstruction. If a carcinoma at the cardia be suspected, a bougie should be passed with great care.

Treatment.—The food should be bland and free from all mechanical irritants. It is thus best to give an aliment of soft semi-fluid or liquid at a temperature not exceeding 100° F. Mastication of any solids permitted should be most complete.

For the hysterical cases treatment by hypnotic suggestion is indicated.

The daily introduction of as large a bougie as possible is very useful. It should be left in situ for about fifteen to twenty minutes, and it is best employed about an hour before food.

Antispasmodics are indicated, and suitable doses of bromides, chloral, belladonna, and carminatives are helpful for all those cases of cardiospasm unassociated with organic disease.

5. Pylorospasm (Spasm of the Pylorus).

The factors associated with pylorospasm are similar to those producing cardiospasm.

All degrees of spasm are met with. There may only be a slight amount, sufficient to cause some delay in the passage of the chyme or the obstruction may be almost complete.

Normally the action of the pylorus is under reflex control, and its function is closely associated with the state of the gastric secretion. Pylorospasm is not an uncommon complication of hyperchlorhydria. In this error of secretion the motor function of the

stomach is unstable, and tends to be over-active. I have never seen pylorospasm in achylia.

Kaudek believes that this affection never occurs apart from a lesion in the stomach wall.

The connexion between the function of the pylorus and the state of the intestine has already been alluded to (*see* page 23). Over-loading of the intestine may reflexly cause closure of the pylorus (pylorospasm), which results in gastric embarrassment and retention. Thus pylorospasm may complicate appendicitis, ileo-cæcal stasis, and enteritis.

Again, the affection may occur in the neurasthenic and hysteric and in such cases both cardiospasm and pylorospasm may be simultaneously present (*see* Pneumatosis, page 316).

Pylorospasm may also arise from local conditions—*e.g.*, duodenitis and peptic ulcer. The ingestion of indigestible food may give rise to gastric irritation sufficient to evoke spasm of the pylorus.

Symptoms and Signs.—The chief features of this condition are regurgitation of gastric contents long after the stomach should have emptied, the belching of wind, pain in the epigastrium, often grinding and spasmodic in character, and at times ' collective vomiting.'

Marked tenderness and rigidity may be made out in the pyloric region by palpation, and gastric succussion, at a time when the stomach should be empty, is frequently elicited.

When the attacks are frequent and prolonged dilatation of the stomach almost always follows.

The diagnosis may be aided by the passage of the stomach tube, when considerable amounts may be withdrawn six to eight hours after food.

X-ray examination is very useful in all doubtful cases, and it may help us to distinguish functional pylorospasm from true pyloric stenosis associated with disease. Clinically it will usually be impossible to differentiate prolonged pylorospasm from organic stenosis without exploration.

Treatment.—If any likely provocative cause can be discovered treatment should be directed to its removal.

If gastric analysis reveals hyperchlorhydria it should be appropriately treated (see hyperchlorhydria). Ileo-cæcal stasis must be overcome by suitable aperients.

The dietary must be carefully regulated and frequent small fluid meals answer best. Switched eggs and curds and cream are

useful. Olive oil is helpful in diminishing over-acidity and in regulating the bowels. Antacids may also be required.

Antispasmodics are indicated in hysteria and neurasthenia, and attention should be paid to the general health. Carminatives, belladonna, rarely opium, bromides, chloretone and chloral hydrate will be found palliative. Adrenalin hypodermically has also been found to give relief.

When the symptoms are urgent the most rapid relief is obtained from the hypodermic injection of $\frac{1}{4}$ grain of morphine and 1/100 of a grain of atropine. If the case be less urgent, this mixture will probably suffice :—

R	Potassii Bromidi	ʒiv.
	Chloretone	ʒiss.
	Tincturæ Belladonnæ	ʒi.
	Syrupi Codeinæ	ʒvi.
	Syrupi Zingiberis	℥i.
	Aquæ Chloroformi.... .... ad	℥vi.
		M.

Sig. :—ʒiv, in water, every four to six hours as may be required.

6. Peristaltic Unrest (Tormina Ventriculi).

This motor neurosis, described by Kussmaul, is of rare occurrence, and it must indeed be difficult of certain diagnosis. It will almost always be found that there is a co-existing lesion to induce the hyper-motility resulting often in visible peristalsis.

I would certainly never diagnose functional ' peristaltic unrest ' unless the absence of lesion was demonstrated by surgical exploration.

7. Aerophagy (Air Swallowing).

Air swallowing is seen most frequently in young hysterical women. It may be voluntary or involuntary, and the condition is largely a bad habit.

Air swallowing is voluntary in diminished intra-gastric pressure, and is thus frequently found as a symptom of myasthenia gastrica, dilatation, and gastroptosis.

There are constant abortive attempts to belch wind till a sufficient amount of air is swallowed. Then a sudden explosive eructation takes place with temporary relief.

TREATMENT.—General hygienic and tonic measures are indicated. The most important part of the treatment is to enlist the co-operation of the patient in a determined effort, and by constant attention, to overcome the bad habit.

Abdominal massage, scientific exercises, and high frequency are also useful measures. Help may also be got from hypnotic suggestion.

Medicinal remedies which promote a better muscular tonus of the stomach may also contribute to improvement. Strychnine and physostigmine may be prescribed in pill, and sometimes I have found carminatives helpful.

8. Pneumatosis.

This disorder is closely associated with neurasthenia and hysteria. When the two gastric orifices become locked at the same time the stomach becomes distended with air.

With each attack there is a sense of gastric fulness and discomfort, and even of actual pain, with abortive attempts at eructation.

TREATMENT.—During the attack the stomach tube should be passed to permit of the escape of gas.

Carminatives, bitter tonics, and antispasmodics are indicated.

Bromide of potassium and occasionally opium may be necessary to give relief.

℞	Spiritus Ammoniæ Aromatici	ʒiv.
	Tincturæ Belladonnæ	ʒi.
	Potassii Bromidi	ʒiv.
	Spiritus Chloroformi	ʒiv.
	Syrupi Zingiberis	ʒi.
	Aquæ Anethi	ad ʒvi.
		M.

Sig. :—ʒiv, in water, to be taken as required.

9. Merycismus (Rumination : Chewing the Cud).

In this condition there is the ability to regurgitate food at will without any nausea or apparent effort. The food is re-chewed and again swallowed.

It occurs chiefly in men, and especially in those of sedentary occupation.

In many instances the affection occurs among the apparently healthy, but it may be associated with neurotic symptoms.

It is supposed that the condition is due to an unnatural relaxation of the cardiac orifice during the digestive act and to the establishment of a habit.

The gastric chemistry reveals no stereotyped alteration in the secretory function of the stomach. There may be hyperacidity, hypoacidity, or even achylia, though the gastric juice may remain normal as well as the motility of the stomach.

Treatment.—As this condition is mostly a bad habit, it can be overcome by attention and determination on the patient's part.

There will be less inducement to return the food to the mouth if after each meal a sufficient dose of hydrochloride of quinine and strychnine is administered to make the gastric contents very disagreeably bitter.

Psychotherapy may also be recommended.

10. Regurgitation.

Is identical to rumination, but the food is not re-swallowed. The same treatment may be adopted as for merycism.

CHAPTER XXIII.

SENSORY DISORDERS.

1. Neurasthenia Gastrica (Nervous Dyspepsia).

The term neurasthenia gastrica is not a good one. Neurasthenia implies a general nerve exhaustion and not a loss of nerve power confined to an individual organ. It is true that various forms of gastric neurosis, with dyspeptic symptoms, commonly accompany general neurasthenic conditions, but the disorder is not confined to the digestive tract.

The term nervous dyspepsia is much more free from objection, for it attempts to describe a condition which is part of a general disturbance.

Nervous dyspepsia embraces a large number of disorders in the sensory, motor, and secretory functions of the stomach. Sometimes the fault is principally confined to one of these ; at other times all three functions may be implicated in the affection.

When only one function is prominently involved the condition has been called *monosymptomatic*, and when several are involved *polysymptomatic* nervous dyspepsia. The latter is far the more common.

It will thus be seen that ' nervous dyspepsia ' includes a vast number of functional digestive derangements and may therefore be considered the commonest ailment we have to deal with.

The following list of ' dyspepsias ' might reasonably come under this head : Gastralgia, gastric hyperæsthenia, bulimia, anorexia nervosa ; almost all the motor disturbances—*e.g.*, Atony, cardiospasm, pylorospasm, gastroptosis, nervous vomiting, etc. ; most of the disorders of secretion—*e.g.*, hyperchlorhydria, gastrosuccorrhœa, achylia, etc.

As quite a number of these conditions present definite clinical pictures, and are of great importance, they have already been separately described under motor and secretory disorders (*q.v.*).

Only those which are specially related to aberration of the sensory function will be dealt with in this chapter.

Etiology of Nervous Dyspepsia.—Under myasthenia gastrica and gastric atony many of the etiological factors of nervous dyspepsia have already been discussed.

All depressing influences, bad hygiene—want of exercise, fresh air and good food; specific fevers and wasting diseases, and every condition which reduces nerve vitality may promote the development of general neurasthenia, of which nervous dyspepsia is usually only a part.

Although the pathology of neurasthenia is not definitely known, one of its commonest and most important manifestations is digestive trouble. These ailments are very prevalent among the Jewish community.

Symptoms.—It will be readily understood that the symptoms of nervous dyspepsia will be as variable as those which characterise general neurasthenia.

There is usually a complaint of constant tiredness and unfitness, with much mental depression and various sensory disturbances. Headaches, inability for mental work, insomnia, cardio-vascular and vaso-motor changes all enter into the picture of nervous dyspepsia.

The special gastro-intestinal symptoms, although variable, are yet sufficiently stereotyped to be more or less characteristic.

Gastric hyperæsthesia after food is a very common complaint. Fulness, weight, and some pain in the epigastric and dorsal regions are commonly experienced after each meal. There may also be present nausea, eructation of wind, vertigo, obstinate constipation, or irregular action of the bowels. Tenderness of the abdomen may be universal or confined to local hyperæsthetic areas.

As in gastralgia, so in nervous dyspepsia, the symptoms have no definite relationship to ingestion nor to the quality or quantity of food taken, but rather appear to be determined by the general condition of the nervous system.

In most instances the victims of nervous dyspepsia have seen many physicians and they come to each doctor with a volume of details, often carefully written out, usually self-contradictory, and they are almost always anxious to express their own views as to the origin and nature of their complaints. The symptoms are painted in most extravagant and exaggerated terms. It is not right,

however, to prejudge such cases on these grounds, and a diagnosis of gastric neurasthenia or nervous dyspepsia should not be arrived at till all other causes have been investigated and excluded. Gastroptosis, gastritis, and peptic ulcer must all be reviewed.

In nervous dyspepsia an investigation of the gastric chemistry is not very informing because the disorders of secretion are so variable. Occult blood ought to be sought for, in the gastric contents and fæces, if ulcer be suspected. Well-marked heterochylia (*q.v.*), lasting over extended periods of time, is all in favour of nervous dyspepsia. While the amount of HCl varies widely there is always a fairly steady secretion of pro-enzyme.

Prognosis.—The prognosis in severe cases of long standing should always be guarded. The minor cases of nervous dyspepsia are usually amenable to proper treatment. The least favourable are those with a pronounced hereditary tendency, but it is most unwise to give a pessimistic opinion to the patient, whose mental attitude is already most lugubrious.

Those cases which are complicated with gastroptosis, gastrectasis, or mucous colitis are most intractable to treatment.

Treatment.—The general condition of the patient should receive most careful attention, and all the health-giving agencies which may be available should be utilized. Fresh air, change of scene, suitable exercise and home physical culture, adequate rest, dietetic measures, general massage, electrotherapy, hydrotherapy, and above all psychotherapy have all their useful place in the treatment of nervous dyspepsia.

All the functions should be regulated. Constipation and the specially prominent gastric symptoms must be dealt with.

In dealing with the victims of nervous dyspepsia a certain amount of firm discipline is necessary, and the physician who can inspire in his patient the fullest confidence in himself will undoubtedly have the greatest success in treatment.

Certain medicinal agents may be indicated. Of these arsenic, strychnine, iron, quinine, phosphorus, cod-oil, glycero-phosphates, hypophosphites, and bitter tonics may be found useful in dealing with certain features of the disease.

If hyper-, hypo- or a-chlorhydria be discovered these disorders of secretion must be appropriately treated by antacids or hydrochloric acid. Some of these prescriptions may be usefully employed :—

℞ Ferri Sulphatis Exsiccati .... gr. ½
Strychninæ
Acidi Arseniosi .... āā gr. $\frac{1}{36}$
Extracti Euonymi Sicci
Extracti Casaræ Sicci .... āā gr. ½
M.

Ft. Pil. Mitte tales 60.
Sig. :—One pill, thrice daily, after food.

℞ Ferri Arsenatis .... gr. $\frac{1}{36}$
Pilulæ Phosphori (B.P.) .... gr. ½
Thyroidei Sicci .... gr. ⅛
Quininæ Hydrobromidi .... gr. 1
Aloini .... gr. ¼
M.

Ft. Pil. Mitte tales 60.
Sig. :—One pill, thrice daily, after food.

℞ Syrupi Hypophosphitis Compositi
Syrupi Ferri Phosphatis c Quininâ et
Strychninâ .... āā ℥iii.
M.

Sig. :—℥i, in water, thrice daily, before food.

℞ Tincturæ Chiratæ
Tincturæ Gentianæ
Tincturæ Cinchonæ Compositæ .... āā ℥i.
Syrupi Glycerophosphatis .... ad ℥vi.
M.

Sig. :—℥ii, in water, thrice daily, before food.

℞ Acidi Nitrohydrochlorici diluti .... ℥iv.
Quininæ Hydrochloridi .... gr. 24.
Tincturæ Nucis Vomicæ .... ℥ii.
Syrupi Aurantii .... ℥i.
Infusi Gentianæ Compositi .... ad ℥vi.
M.

Sig. :—℥iv, in water, thrice daily, before or after food.

When HCl is diminished this mixture may be given after food. When it is in excess it may be given before food or the mixture may be used as an appetiser.

℞ Magnesii Carbonatis Ponderosi
Bismuthi Carbonatis .... āā ℥vi.
Spiritus Ammoniæ Aromatici .... ℥iv.
Tincturæ Belladonnæ .... ℥i.
Tincturæ Cardamomi Compositæ .... ℥vi.
Aquæ Chloroformi.... .... ad ℨvi.
M.

Sig. :—℥iv, in water, thrice daily, fifteen minutes after food. (As antacid.)

℞ Olei Phosphorati (B.P.) .... m 12—24.
Olei Morrhuæ .... ad ℨvi.
M.

Sig. :—℥ii to iv, thrice daily, after food.

From their composition the physician will easily discern the appropriate use of these formulæ in combating the various aberrations incident to nervous dyspepsia.

2. Hyperæsthesia Gastrica.

By hyperæsthesia gastrica is meant a hypersensitiveness of the gastric mucosa even to its normal contents. The condition may be regarded as a supersensitiveness of the nerve endings in the mucous membrane of the stomach, or more probably, as Hurst has pointed out, in the sub-peritoneal tissue.

It may be a condition *per se* without obvious lesion, and it is this form of the disorder which is here described.

But abnormal sensitiveness of the gastric mucous membrane is also an accompaniment of almost all the serious organic diseases—gastritis, ulcer, and carcinoma, as well as of certain disorders of secretion.

In the purely neurotic form of hyperæsthesia gastrica the patient becomes conscious of the digestive process, and when the food comes into contact with the mucosa it gives rise to discomfort or pain.

This malady is very closely allied to gastralgia.

Etiology.—The same etiological factors are in operation in hyperæsthesia gastrica as in gastralgia.

It may be associated with organic disease of the central nervous system or with various functional neurosis—*e.g.*, hysteria and

neurasthenia. All debilitating conditions tend to induce the abnormality. It is particularly common in young women, and especially in those suffering from *chlorosis*. In former years it was a familiar ailment in my wards in the Western Infirmary, but more recently it has become a rare condition. Either chlorosis is now occurring in Glasgow quite infrequently or few cases are seeking advice at our hospital.

Symptoms.—Unlike gastralgia the symptoms are closely related to the ingestion of solids or fluids.

The pain which occurs is far less severe than in gastralgia. It is better described as a sense of weight, fulness, and discomfort after food than an actual pain.

These symptoms may culminate in nausea or vomiting. Pungent fluids, alcohol and ærated beverages are particularly prone to cause irritation, but otherwise the amount and nature of the food has little influence on the symptoms.

If the condition is not treated the symptoms become aggravated and resemble those of super-secretion, although a gastric analysis may show that the discomfort is arising from a gastric juice of normal acidity acting on a supersensitive mucosa.

As a rule the appetite remains good, although to avoid discomfort the patient eats less than he desires to do. The motor function of the stomach remains intact.

In hyperæsthesia gastrica, of a persistent character, before we accept a diagnosis of this functional neurosis, organic lesion must be excluded. Here again the therapeutic test will assist in excluding ulcer. If relief does not rapidly follow appropriate treatment for ulcer we are probably dealing with hyperæsthesia gastrica.

Treatment.—The diet should be bland and at the outset of treatment principally fluid. Milk and lime water, Horlick's milk, Benger's food, Allen and Hanbury's diets, Ovaltine, egg switch, and jellies are all suitable, and a meal can be given at two hourly intervals.

Treatment should be directed to overcome constitutional disturbance and digestive derangement.

The constitutional treatment may include all the measures narrated for the relief of gastralgia (*q.v.*).

If chlorosis exists the hypodermic use of iron and arsenic (Molteni's preparation, *q.v.*) to avoid any risk of gastric irritation will be found to give excellent results.

Massage, passive exercises and high frequency current may be found helpful for those specially debilitated.

A large poultice of 'mustard bran' placed over the gastric region, and kept there all night, I have found very soothing.

Medicinally gastric sedatives are indicated.

The use of local anæsthetics, such as cocaine or eucaine, although giving quick temporary relief, should be forbidden, as they are almost certain to create a 'habit.' When the symptoms are urgent a few grains of orthoform may be given before meals to diminish the discomfort of the digestive act. This anæsthetic may be used with less objection.

Small doses of belladonna, conium, aconite, carbolic acid, menthol and carminatives are all safe and useful in proper doses.

℞	Tincturæ Belladonnæ	℥i.
	Tincturæ Aconiti	m24.
	Tincturæ Conii	℥i.
	Acidi Carbolici Liquefacti	m12.
	Syrupi Aurantii	℥i.
	Aquæ Carui	ad ℨvi.
		M.

Sig. :—℥iv, in water, every three or four hours, as may be required, before food.

℞	Liquoris Bismuthi	ℨiii.
	Acidi Hydrocyanici diluti	m48.
	Syrupi Codeinæ	℥iv.
	Liquoris Magnesii Carbonatis	ad ℨvi.
		M.

Sig. :—℥iv, in water, before food.

Spa treatment and psychotherapy have also their place in the management of hyperæsthesia gastrica.

3. Gastralgia Nervosa.

This affection has been variously designated as cardialgia, gastric neuralgia, and gastrodynia.

Gastralgia is characterised by the occurrence of intermittent painful seizures which bear no relation to the periods of functional activity of the stomach.

The affection occurs chiefly in women, and for variable intervals there may be complete immunity between the seizures.

Etiology.—Gastralgia may be regarded as a neuralgia of the gastric terminations of the vagus nerve.

It may originate in a variety of ways.

(1) It may take origin in the stomach itself. Improper or excessive food, over-use of alcohol and tobacco and other toxic agents may be directly causative with or without actual gastritis.

(2) It may be a feature of organic disease of the central nervous system. A very severe form occurs in tabes and in some cases of myelitis.

(3) It is not uncommon as a complication of functional derangements of the nervous system. We have examples of it in neurasthenia and hysteria.

(4) It may arise reflexly from disease of other abdominal or pelvic organs. It is thus an intercurrent affection in disease of the uterus and ovaries. Most frequently, however, gastric pain due to reflex irritation is of the nature of a spasm rather than a neuralgia.

Symptoms.—The attacks are characterised by sudden onset, with or without nausea or vomiting. Noisy eructations of wind are frequent.

The pain may be slight or severe. The slight attacks pass over in an hour or two, but the severe seizures may persist for some hours to a few days.

In the severe cases the pain reaches its maximum in a few minutes and is principally situated in the epigastrium and left hypochondriac region. From these situations it may radiate over a wide area and spread up the left side to the left shoulder and through to the back.

Associated with the pain there may be pronounced cardio-vascular disturbance with faintness, pallor, coldness of the extremities, subnormal temperature and arythmia.

The patient usually assumes a doubled-up posture with his knees pressed against his chest. Doubtless this decubitus tends to promote abdominal relaxation. The face takes on an anxious expression and is often bedewed with sweat.

When the attack passes over the patient is somewhat exhausted and may fall asleep.

Physical signs are not definite in gastralgia. There may be tenderness in the gastric region and some abdominal retraction.

Differential Diagnosis.—It will be seen from the above clinical picture that an attack of gastralgia is not dissimilar to one of *renal* or biliary colic, hyperchlorhydria with pylorospasm, peptic ulcer, intestinal colic, or true or false angina pectoris.

A thorough investigation of the case and a consideration of the history will aid the diagnosis.

The attacks are usually of short duration and show no definite periodicity. Jaundice is absent and gall stones will not be found in the fæces. The absence of urinary symptoms will usually exclude renal colic, and a normal heart and blood pressure are against angina pectoris. A chemical examination of the gastric contents giving a normal gastric secretion will enable us to negative hyperchlorhydria and pylorospasm.

Ulcer may be discounted by the therapeutic test. Gastralgia yields quickly to treatment and the symptoms remain in abeyance while in peptic ulcer relief is only temporary. Chemical examination of the gastric contents and fæces for HCl and occult blood, respectively, will also aid the diagnosis.

Treatment.—Prophylactic treatment can only be initiated if we are able to discover the causative factors of the gastralgia.

If there be any dietetic error it should be corrected. The over-indulgence in alcohol or tobacco should be stopped, and the general health should be improved and neurasthenic symptoms dealt with.

The treatment of the actual attack will depend on its severity. No food should be given, but a drink of hot water containing a carminative I have found useful in relieving the pain of the milder cases. This carminative acts well :—

℞ Spiritūs Myristicæ
Spiritūs Cajuputi ....
Spiritūs Chloroformi
Spiritūs Menthæ Piperitæ .... āā ℥i.
M.

Sig. :—Half to one teaspoonful, in a tumblerful of hot water, to be repeated as may be required.

Warm applications to the stomach should always have a trial. If no improvement follows these measures in a reasonable time then

we ought to prescribe sedatives. Before giving opiates the following mixture may be tried :—

℞ Potassii Bromidi .... gr. 60.
Syrupi Chloral .... ʒiii.
Spiritus Chloroformi
Spiritus Ammoniæ Aromatici .... āā ʒii.
Aquæ Menthæ Piperitæ .... ad ℥iii.
M.

Sig. :—One tablespoonful, every two hours, till pain be relieved.

Or ten grains of phenazone or phenacetin may be preferred. These drugs usually suffice to give relief in the milder cases.

For those which are very acute and associated with urgent symptoms the best remedy is a hypodermic of morphine and atropine (gr. $\frac{1}{4}$ and 1/100), or gr. 1/12 of heroin hydrochloride.

When it can be obtained a strong galvanic current of three to five milliamperes, with the anode on the stomach and the cathode on the dorsal spinal region often gives rapid relief. The current should be allowed to flow for five to ten minutes and be slowly reduced before the electrodes are removed.

A course of arsenic, in the form of Fowler's solution, two to three minutes before food, gives excellent results. It may be combined with a bitter and carminative :—

℞ Liquoris Arsenicalis .... ʒi.
Tincturæ Chiratæ
Tincturæ Cardamomi Compositæ āā ʒvi.
Aquæ Chloroformi.... .... ad ℥vi.
M.

Sig. :—ʒii, in water, thrice daily, before food.

4. Bulimia (Ox-hunger, Cynorexia, Lycorexia, Hyperorexia).

Bulimia may be defined as an abnormal craving for food with an insatiable appetite. True bulimia should not be confused with the symptomatic hunger occasionally met with in hyperchlorhydria and peptic ulcer, diabetes, Basedow's disease, tœnia, and some other conditions.

The pathogenesis of the abnormality is not exactly known, although many explanatory theories have been advanced.

Anæsthesia of the endings of the peripheral nerves of the stomach, contraction of the gastric muscularis and irritation of the hunger centre in the medulla have all been given as causative factors.

It is clear that certain diseases of the central nervous system may give rise to bulimia. A case of acromegaly which I had under observation in my wards in the Western Infirmary for a period of two years, from time to time, developed an extreme bulimia with a wolfish appetite. He had to be kept under observation during meal hours or he would find his way to the ward kitchen and eat the rations assigned to other patients.

Symptoms.—The condition occurs principally in women. The abnormal appetite and sense of gastric emptiness may be continuous, but much more commonly the attacks are intermittent and periodic.

Various subjective symptoms accompany the condition—vertigo, faintness, coldness of the extremities and gnawing pain in the stomach. The amount of food taken is enormous and is eaten voraciously.

Examination of the secretory and motor functions of the stomach usually reveals no abnormality.

Treatment.—If any abnormality can be discovered which is likely to be provocative it should, if possible, be removed.

In the neurotic cases, without discoverable cause, the patient should be fed hourly, and if possible be compelled to eat slowly. The latter condition may be promoted by separating the solids from the fluids. Psychotherapy should be employed. Medicinally belladonna, bromides and arsenic are useful. Very small doses of codeine may be employed for severe intermittent attacks, but this drug should not be administered for long periods of time. The undernoted formula answers well :—

℞ Tincturæ Belladonnæ	....	℥ii.
Potassii Bromidi	....	℥iv.
Liquoris Arsenicalis	....	m48.
Syrupi Codeinæ	....	℥vi.
Aquæ Chloriformi	....	ad ℨvi.
		M.

Sig. :—One tablespoonful, in water, as may be required, before food.

5. Polyphagia.—This affection is closely allied to bulimia. It is a paroxysmal trouble associated with intense hunger when the digestive act is over and the stomach empties. The periods over which the attacks last are very variable. The over-eating may lead to acute or chronic dilatation. The condition requires the same management and treatment as bulimia.

6. Sitisphobia is a morbid fear of eating. The abnormality arises from idiosyncrasy, fastidiousness and prejudice with regard to certain articles of food. It may develop to such an extent that one article of diet after another may be refused till the patient will almost take nothing, and anorexia and emaciation supervene.

The treatment which is most indicated is a complete rest cure (Weir Mitchell) and psychotherapy.

7. Parorexia.—This condition may be defined as a perversion of appetite with a craving for unusual foods or for non-alimentary substances.

Highly spiced foods—mustard, sour things, unripe fruits; uneedible substances—coal, ashes, mud; and disgusting or dangerous things—menstrual secretion, urine, fæces, needles, glass, may form the objects of craving in sufferers from parorexia.

Treatment should be by discipline and psychotherapy.

8. Anorexia Nervosa.—Anorexia nervosa occurs in young hysterical subjects. It may be defined as a loss of appetite often with a distinct aversion to food. This condition is ascribed to a depression of the hunger centre or hyperæsthesia of the gastric mucosa. A symptomatic anorexia may be due to the excessive use of tobacco or to the abuse of opium.

Symptoms.—The loss of appetite comes on gradually. It may, however, be sudden on its onset, following some severe emotion or shock. The trouble may begin with a distaste for certain articles of diet until all become objectionable to the patient. Nutrition gradually fails and the patient becomes more nervous and anæmic. In severe conditions the signs and symptoms of starvation may ultimately supervene.

Treatment.—The neurotic element should be treated by a rest cure (Weir Mitchell), general massage, and high frequency current.

Bitter tonics and hydrochloric acid are also helpful. Iron and arsenic should be administered if anæmia exists.

℞ Acidi Hydrochlorici diluti .... ℥iv.
Glycerini Pepsini .... ℥ii.
Liquoris Arsenici Hydrochlorici .... ℥ii.
Quininæ Hydrochloridi .... gr. 24.
Infusi Gentianæ Compositi .... ad ℥vi.
M.

Sig. :—℥iv, in water, thrice daily, ten minutes before food.

℞ Tincturæ Cinchonæ Compositæ .... ℥vi.
Liquoris Strychninæ .... ℥i.
Tincturæ Capsici .... m30.
Syrupi Limonis .... ℥i.
Infusi Gentianæ Compositi .... ad ℥vi.
M.

Sig. : —℥iv, in water, thrice daily, before food.

In most instances there is a pronounced neurotic temperament behind this condition.

When nutrition threatens to fail lavage may advantageously be carried out, and when all the fluid is returned an abundant nutrient meal of eggs, milk, and extractives should be passed into the stomach before the tube is withdrawn.

SECTION IV.

STOMACH SYMPTOMS IN RELATION TO DISEASES OF OTHER ORGANS.

CHAPTER XXIV.

DISEASES OF PANCREAS, GALL BLADDER AND BOWEL

1. Diseases of the Pancreas.

The pancreas is liable to many primary and secondary diseases of an acute and chronic type. Only the more important of these can be mentioned here. A detailed description is outside the scope of this work. When the pancreas, perhaps one of the most important organs in the body, becomes the seat of disease it produces widespread disorder. Diseases of the gland are so commonly associated with digestive derangements, the true cause of which is often overlooked, that it is essential to give this subject some consideration. The gland has such important digestive functions that when it is efficient it is almost possible to dispense with gastric digestion. The difficulty in diagnosing disease of the pancreas arises from its great complexity of function and the diversity and variability of the symptoms which accompany its disorders. The diagnostic fat necrosis of the gland is only demonstrable during life at the laparotomy, or at the *post mortem* examination. Most frequently the symptoms might well be those of disease of other parts of the digestive tract, and indeed at the outset they are usually ascribed to gastric derangement. Thus when pancreatic disorder is suspected we must invoke all the possible laboratory aid to estimate if the functional activity of the gland is abnormal or suppressed.

The pancreatic gland has two great functions to perform :

(1) It elaborates an external digestive secretion containing ferments which can digest, in an alkaline medium, all classes of aliment (*see* physiology of digestion).

(2) It elaborates an internal secretion, derived from the specialised glandular tissue known as the islands of Langerhans, which has a controlling influence on carbohydrate metabolism.

When the gland is extirpated, or becomes sufficiently diseased to destroy the acini of the islets of Langerhans, and the internal secretion ceases to be formed in sufficient quantities, the close inter-relationship between this secretion and that of the adrenals is vitiated and diabetes mellitus results.

Of the primary lesions of the gland the most common are acute and chronic pancreatitis, cancer, cysts, and calculi.

Acute Hæmorrhagic Pancreatitis.—This disease is rare in youth. It is most common in stout people who are at, or have passed, middle life. The onset has a dramatic suddenness and is associated with the most pronounced symptoms. The clinical picture is that of the 'acute abdomen' of an exaggerated type, and most commonly acute pancreatitis is diagnosed as perforated gastric ulcer, ruptured appendix, or biliary or renal colic. It is not uncommonly associated with cholecystitis.

Symptoms.—The disease is ushered in by nausea, quickly followed by violent and persistent vomiting.

Severe pain is an early and prominent symptom. It may be continuous or paroxysmal, and is chiefly complained of in the back and left gastric region.

There is great rigidity of the abdominal wall and the colon is distended. Slight cyanosis is rather typical of the syndrome. Collapse is pronounced. These symptoms follow each other in rapid succession.

Constipation may be present, but frequently there is diarrhœa.

A little later jaundice ensues in the majority of cases. This is due to pressure on the bile duct, which in the great majority of cases (70 per cent.) passes through the head of the gland and so becomes implicated. The motions are pale even though jaundice be absent.

Sugar appears in the urine. The blood shows a slight leucocytosis.

The acute inflammatory process may proceed to suppuration or gangrene. At this stage tumour-formation may be made out projecting into the epigastrium. When these changes occur in the gland rigors and irregular pyrexia are added to the symptoms, and the blood shows an increased leucocytosis. The facial expression becomes hippocratic and the condition makes rapid progress to a fatal termination.

As the clinical features of this disease are not sufficiently characteristic to make the diagnosis always certain we must seek further aid from certain tests.

(*a*) The Loewi eye test can be quickly carried out by dropping into one eye, at short intervals, a few drops of a 1 in 1,000 solution of adrenalin. Mydriasis is a positive reaction indicative of pancreatitis.

(*b*) The urine should be examined for excess of diastase, which is usually marked in the early stages of the disease.

It should also be tested for sugar, as glycosuria is characteristic and examined for crystals of calcium oxalate.

(*c*) The fæces should be examined for occult blood. In almost every case of acute pancreatitis this is present.

Although all these tests are very useful signs of acute pancreatitis, when positive, events march so quickly that if the disease be strongly suspected from the clinical features a laparotomy ought to be performed at the earliest moment. Undue delay seriously compromises the life of the patient. No medical treatment is of any avail. The abdominal cavity will be found to contain blood-stained fluid and the typical spots of fat necrosis may be seen.

Chronic Pancreatitis.—The chronic type of this disease is much more insidious and consequently more difficult of recognition. The abdominal disturbance is slight and the symptoms are usually assigned to some chronic gastric affection.

Chronic pancreatitis is closely related to cholelithiasis, cholecystitis, chronic peptic ulcer—especially duodenal—and pancreatic calculi.

The liability to pancreatitis, from these affections, in different individuals, is probably determined by the exact relationship which exists between the pancreatic and common bile ducts. When the two enter the duodenum together infections from the bile duct are much more liable to spread to the pancreas than when the ducts enter separately.

Symptoms.—The main features are debility and general malnutrition expressed by progressive loss of weight. A palpable tumour may be felt.

Added to these there may be nausea, vomiting, epigastric pain, sallow complexion and pigmentation of the skin, large fatty stools (creatorrhœa and statorrhœa), glycosuria and certain other abnormalities in the urine and fæces to be ascertained by testing.

Such symptoms, following a history of chronic cholelithiasis or chronic duodenal ulcer, should always cause an investigation of the functions of the pancreas to be undertaken.

If and when the bile duct becomes compressed, from an interstitial pancreatitis, jaundice may slowly develop.

In those cases (30 per cent.) in which the duct passes behind the pancreas jaundice may be absent.

Evidence of interference with the external or internal secretion is likely to develop in course of time.

The stools may become bulky and fatty. These changes are due to the failure of intestinal digestion of the proteids and fats. The starches are usually well digested, which suggests that there are other ferments which supplement the diastatic action of the normal pancreatic secretion. No doubt the succus entericus has an important action in supplementing pancreatic digestion.

Steatorrhœa, or the evacuation of stools containing liquid fat, which solidifies on cooling, is a very diagnostic feature of pancreatitis if associated with other symptoms and signs.

What is most characteristic is the defective amount of split fats (fatty acid and soaps). Normally, of the fats excreted, 80 per cent. should be in this form, but in chronic pancreatic disease they may be reduced to about 20 per cent. This absence of lipolysis is practically diagnostic of pancreatic failure.

Creatorrhœa is commonly combined with steatorrhœa. By creatorrhœa is meant the evacuation of unchanged proteins. The microscope is the best evidence of the presence of the condition. Muscle fibres with their histological structure preserved are very typical of failure of pancreatic digestion. Corroboration of this failure may be obtained by the administration of commercial stained nuclei and their detection in the fæces unchanged by digestive action.

In health the external pancreatic secretion is formed in excess of the amount required to complete the digestion of a mixed meal. Thus a fæcal extract should contain a surplus which is capable of digesting a certain amount of cooked starch which may be added to it.

(1) Make a 10 per cent. solution of the fæces in distilled water, by mixing well in a mortar, and allow it to settle for two hours.

(2) Then filter repeatedly till a quite clear solution be obtained.

(3) To six test tubes add :—

2 c.c., 1.5 c.c., 1 c.c., 0.5 c.c., 0.1 c.c., 0 c.c.

(4) Make a 1 per cent. solution of starch by just bringing the water to the boiling point. Allow it to stand till it has a temperature of 100° F., and then add to each tube 5 c.c. Shake well.

(5) To each tube add one drop of toluol to act as an antiseptic, and inhibit fermentative action by micro-organisms. Shake well. Plug the mouth of each test tube with a pledget of cotton wool.

(6) Incubate, at 37 C, for twenty-four hours.

(7) Add distilled water to each tube till about three-quarters full. Shake well.

(8) Add to each one drop of decinormal solution of iodine. Note the colour developed in each tube in a good light :

Blue colour.—Starch undigested (dextrin).

Burgundy colour.—Starch partially digested (erythrodextrin).

No change in colour.—Starch well digested (achroödextrin).

The fourth tube, containing only 0.5 c.c. of fæcal extract, is so attenuated that it can only partially digest the added starch. The burgundy colour of erythrodextrin is therefore normal in it.

The first three tubes should remain colourless, because when the pancreatic secretion is abundant the starch in these is completely digested.

The sixth tube, with no fæcal extract, is used as a control. It stains a deep blue or blue-black from unaltered starch.

Diastase.—This ferment is present in variable quantities in the blood of the healthy subject, and is excreted proportionately in the urine.

In diseases of the pancreas with failure of secretion diastase becomes greatly increased. The greater the amount found, the more certainly is pancreatic disease indicated.

The test for diastase is carried out in a similar way to the above, and the estimation is made by the capacity the urine has to digest a given quantity of a standard solution of starch. A 1/50 normal solution of iodine is used as the indicator.

Sugar tolerance test.—In pancreatic insufficiency the sugar tolerance is greatly reduced. The normal subject should be able to take 100 grams of glucose on an empty stomach and milk diet without the production of a temporary glycosuria. If the pancreas is diseased the tolerance may be reduced to 25 to 50, or at least below 100 grams.

Einhorn's duodenal tube.—There can be little doubt that the most direct way to interrogate the functions of the pancreas is by Einhorn's duodenal tube. Through it the duodenal contents can be aspirated and examined for the pancreatic ferments.

Pancreatic Carcinoma.—Carcinoma of the pancreas is difficult to diagnose in its early stages. The implication of the biliary duct in the growth may cause early and persistent jaundice, which is apt to be ascribed to hepatic disease and its pancreatic origin overlooked.

The signs and symptoms associated with chronic pancreatic disease are likely to develop and later on definite tumour. Physical deterioration is very rapid.

Pancreatic Cysts.—Small cysts escape notice during life. The larger ones are palpable in the epigastric region as elastic rounded tumours. Sometimes fluctuation may be made out if the abdominal wall is not too thick.

If the tumour or tumours be large, pressure on the contiguous organs may give rise to additional symptoms. Glycosuria is usually intermittent, but all the symptoms of chronic pancreatitis may be present.

Pancreatic Calculi.—The main symptom is pain in the epigastrium radiating to the left side or up to the left shoulder.

During the passage of a calculus the pain is severe and associated with vomiting, passing jaundice, glycosuria, bulky fatty stools (steatorrhœa and creatorrhœa).

The treatment of almost all of these pancreatic diseases is surgical. Medical treatment need not, however, be neglected. It may prove beneficial in aiding intestinal digestion and palliating certain symptoms of chronic pancreatitis.

Fortunately carbohydrate digestion is not completely in abeyance even though pancreatic disease exists. By insisting on prolonged mastication, to ensure thorough insalivation, the ptyalin may supplement to some extent the defect in pancreatic secretion. Later on the starches are acted on by the succus entericus.

The inability to digest fats suggests that they should be strictly limited. They can be utilised to some extent if they be well emulsified. Virol and cod oil and malt extract may be given in small doses to aid nutrition.

One might expect pancreatic preparations to be helpful, but I have not found them specially useful.

Duodenal alimentation by means of Einhorn's tube promises to give good results. Predigested foods can be directly introduced into the duodenum and interference with the stomach avoided.

Of medicinal agents, salicylic acid, aspirin, helmitol, are supposed to find their way to the pancreas and act as local disinfectants. One or other of these drugs may be given in gr. 10 doses after food. The drug which I have found most useful, as a disinfectant for both gall-bladder and pancreas, is saliformin (Merck). This preparation is a combination of acidum salicylicum and formaldehyde. It may be given in doses of 7 to 10 grains thrice daily after food.

Various intestinal antiseptics to diminish auto-intoxication may also be employed. Hydrochloric acid and bitters may improve the appetite and indirectly stimulate the secretory function of the pancreas.

2. Cholelithiasis (Gall Stones).

Gall-stones owe their origin principally to the action of micro-organisms and to altered conditions of the bile. The gall-bladder is an excellent incubating chamber. Nearly all the organisms met with in the bowel have been found in it, and the enteric bacillus has been recovered, in pure culture, as long as twenty years after an attack of typhoid. It is thus that certain people, apparently otherwise in perfect health, may be 'carriers' and originate an epidemic of this disease.

Gall-stones almost always give rise to symptoms of chronic dyspepsia, and even when jaundice is a feature of the condition the true cause of the gastric derangement is apt to be overlooked and ascribed to peptic ulcer (duodenal) or to gastritis. The absence of jaundice should not exclude the possibility of cholelithiasis.

Sooner or later the presence of gall-stones is apt to set up irritation in the gall-bladder and a complicating cholecystitis may supervene which aggravates the symptoms and local signs.

Other complications may occur. The stone may become impacted in the common bile duct with deep and permanent jaundice, or it may ulcerate its way into the bowel, especially the first part of the duodenum, and be passed per rectum.

If the cystic duct becomes the seat of obstruction by gall-stones, enormous dilatation of the gall-bladder may occur. If the obstruction be sudden, the fluid which gathers is bile-stained, but when the obstruction is chronic, exploratory aspiration of the elastic tumour will reveal a clear mucoid fluid.

The dyspepsia of cholelithiasis may be described as reflex, but if cholecystitis complicates the condition adhesions may form between the gall-bladder and the duodenum, which may directly interfere with the function of the stomach.

The symptoms of cholelithiasis depend greatly on the situation of the calculus or calculi and on their size. If only one large calculus be present in the gall-bladder there may be no biliary colic and no jaundice. But if the gall-bladder contain numerous small calculi, one or more of them may find their way into the bile duct at irregular intervals and produce the acute symptoms of biliary colic.

Cholelithiasis occurs in older people than duodenal ulcer, and it is most common in stout women over forty years of age.

In chronic cholelithiasis, even when there are no attacks of biliary colic, symptoms of dyspepsia of an intermittent and irregular character are commonly experienced.

Nausea and vomiting without apparent cause are frequent symptoms. The vomiting has no definite relationship to ingestion. It is associated with pronounced flatulence and belching, and there may be palpitation and dyspnœa.

Pain is usually complained of in the epigastric region and it is aggravated by lying on the left side, because the gall-bladder drags on its congested peritoneal ligament. Like the vomiting, the pain has no close connexion with eating and it is not relieved by vomiting or by alkaline mixtures. According to Moynihan, during an acute attack of pain there may be spasm of the diaphragm which momentarily causes a characteristic catch in the breathing.

The pallor of the stools will vary with the depth of the jaundice. Slight icterus following an attack of acute pain is in favour of cholelithiasis. A calculus lying at the ampulla of Vater and acting like a ball-valve is commonly associated with irregular attacks of icterus, shivering and slight elevation of temperature (hepatic intermittent fever of Charcot).

After each attack of biliary colic the motions should be sieved and gall-stones sought for. The stools should be kept under observation for at least four days. If gall-stones are found their appearance should be carefully studied. When facetted we know that many are present in the gall-bladder, while if rounded and eroded we may conclude that there are no more, or at least that they are not numerous.

Examination of the gastric contents shows well-marked hyperchlorhydria in the majority of cases. There is usually much congestion of the gastric mucous membrane, and if retching be violent minute quantities of occult blood may sometines be found in the gastric contents. In such circumstances the differential diagnosis of cholelithiasis and duodenal ulcer becomes increasingly difficult. Fortunately it is not common for macroscopic or occult blood to occur in the vomitus or fæces in cases of cholelithiasis. The urine may be bile-stained and it may contain urobilin.

Palpation of the abdomen may reveal some tenderness, fulness and rigidity in the region of the gall-bladder and Reidel's lobe (a tongue-like projection of the right hepatic lobe) may be displaced downwards.

Tenderness may be elicited by deep pressure on the right side of the vertebral column from the tenth dorsal to the first lumbar vertebræ.

Chronic cholelithiasis and cholecystitis, if untreated, are the commonest causes of pancreatitis.

3. Cholecystitis.

Inflammation of the gall-bladder may occur with or without gall stones. While it is a very common concomitant of cholelithiasis it may occur primarily from the action of various toxins, especially from the bacterial infection of enteric fever.

Cholecystitis occurs as an acute or chronic affection. Sometimes the latter is a sequel to the former. In the acute types the toxic action may be so severe that suppuration (empyema of gall-bladder) or gangrene takes place.

Chronic cholecystitis is most frequently associated with gall-stones. The symptoms are milder and very apt to be ascribed to gastric derangement.

There is very little difference in the clinical features of cholelithiasis and cholecystitis.

In the acute disease the digestive derangement is pronounced and is associated with constitutional disturbance. Rigors, fever, severe pain and vomiting may all be in evidence. Jaundice is absent in most instances of uncomplicated cholecystitis.

There may be marked pain, tenderness, and sometimes rigidity over the gall-bladder, which may be felt to be very tense and much distended.

The diagnosis between cholecystitis and cholelithiasis is often very difficult to make, and the physician will have to differentiate both conditions from duodenal ulcer and from hyperchlorhydria. Only by carefully weighing the evidence obtained by all methods of investigation can we hope to arrive at a probable diagnosis. An X-ray investigation is often very helpful in excluding duodenal ulcer, although it rarely gives positive evidence of the presence of gall stones.

4. Intestinal Disorders.

Dyspeptic symptoms, often apparently referable to the stomach, take their origin in various disorders of the intestinal tract. Of these only a few of the more important can be dealt with here.

(*a*) **Intestinal obstructions.**—All forms of acute and chronic obstructions may give rise to digestive derangements which may be confused with primary gastric disorders.

In *acute obstructions* there is usually little difficulty in recognising the nature of the condition. Persistent and intractable vomiting, ultimately becoming fæcal in character, is the leading feature of the disease. Later on, local or general abdominal distension is added to the picture.

The *chronic progressive obstructions* are more difficult of diagnosis because the symptoms are less pronounced. All those which we commonly associate with gastric dyspepsias may be the only ones complained of. Abdominal uneasiness, flatulent distension, constipation or irregular action of the bowels, nausea, vomiting, and a variable amount of pain may be usual symptoms of abdominal obstruction and of many common gastric affections.

Generally speaking pain below the umbilicus may be ascribed to the bowel and that above it to the stomach, pancreas, or gall-bladder.

(*b*) **Appendicitis.**—*Acute appendicitis* is not commonly difficult of diagnosis as the symptoms and local signs are so pronounced and typical. If perforation occurs there may be difficulty in the differential diagnosis from perforated gastric ulcer, but as both conditions indicate immediate laparotomy an error of this kind is not of serious consequence.

Chronic appendicitis.—By far the more important of the intestinal conditions giving rise to digestive derangements, simulating gastric disorders, is chronic appendicitis. There is no abdominal

disease in which we get a greater variation of symptoms and signs. It is quite common to find that there is little to call attention to the appendix and much to direct it to the stomach. The connexion between the appendix and the duodenum has already been referred to, and it is not an uncommon occurrence to find both simultaneously the seat of disease.

The disease with which chronic appendicitis is most likely to be confused is duodenal ulcer, but it may also simulate cholelithiasis and cholecystitis.

There may be nausea, vomiting after food, marked epigastric pain and hyperchlorhydria. There may be no alteration in the pulse or temperature. There is usually a history of constipation.

The pain of chronic appendicitis is usually below the umbilicus while that of duodenal ulcer is above it, and it has not the definite relation to ingestion which is typically connected with duodenal ulcer.

In chronic appendicitis the pain radiates to the right rather than to the left side, as it does in duodenal ulcer.

There is often marked tenderness in appendicitis at McBurney's point with rigidity, sometimes fulness and dulness on percussion. But there are so many atypical cases in which all the diagnostic rules are upset that the absence of these typical signs should never mislead us.

Rectal or vaginal examination may be more informing, in cases of difficulty, than external abdominal examination.

Hyperchlorhydria is frequently present in chronic appendical dyspepsia as it is also in duodenal ulcer, but it is rare to find occult blood in the fæces in uncomplicated appendicitis.

In every case of suspected duodenal ulcer the appendix ought to be carefully examined and disease of that organ excluded.

CHAPTER XXV.

DYSPEPSIAS OF PULMONARY, CARDIAC, TOXIC AND BLOOD DISEASES.

5. The Dyspepsias of Pulmonary Affections.

(*a*) **The Dyspepsia of Phthisis.**—It is not uncommon for the preclinical stage of pulmonary phthisis to be associated with gastric derangement. Tuberculous gastritis, from infection by the swallowed sputum, is comparatively rare. The dyspepsia of phthisis is of a functional character.

Symptoms.—The appetite becomes impaired. There is a sense of satiety after a little is taken, and indeed there may be complete anorexia. Discomfort and fulness are complained of in the epigastrium, and there is usually a good deal of flatulent eructation. The tongue is flabby, often too red, and covered with an irregular coating. Nausea is frequent, but vomiting is not common except when it occurs as a complication of violent coughing.

The gastric chemistry is very variable. It has been noted by some observers that in acute cases with fever there is usually a subacidity or even a total absence of HCl. In those cases without fever the gastric secretion may be normal. Sometimes heterochylia is well marked. As the disease advances and debility and emaciation progress both the secretory and motor functions of the stomach tend to diminish. As a late manifestation considerable dilatation may be found.

In every case of apparently causeless dyspepsia in the young the chest ought to be kept under observation, clinically and by X-ray, to exclude pulmonary disease. According to Hausmann the diagnosis of suspected incipient phthisis may be confirmed, before other symptoms develop, by finding tubercle bacilli, which have been swallowed during sleep, in the contents of the fasting stomach.

The *prognosis* is dependent on the chest condition.

Treatment of the dyspepsia of phthisis is that of chronic gastritis. In spite of the bad appetite a persistent attempt at forced feeding must be maintained and all the hygienic measures usually adopted for the treatment of phthisis brought into operation.

Bitter tonics, strychnine, hydrochloric acid, syrup of hypophosphites, etc., may prove useful in improving the appetite. If hyperchlorhydria should be a feature of the case alkalies are indicated.

(*b*) **Asthma and Chronic Bronchitis with Emphysema** are commonly associated with dyspepsia. In any condition of the lungs and heart which produces chronic venous congestion (emphysema and valvular disease with failing compensation) all the symptoms of chronic gastritis may be present.

6. Cardiac Disease.

It has already been noted that in many gastric affections reflex disordered action of the heart is quite common. Both arhythmia and tachycardia are met with, especially when there is flatulent distension or retention of the gastric contents.

But, on the other hand, disorder and disease, primary to the heart, may induce serious derangement of the functions of the gastro-intestinal tract and liver.

Whenever the myocardium is the seat of degeneration, whether primary or secondary to valvular lesions, and failure of compensation arises, the portal circulation sooner or later becomes interfered with and general venous congestion of the abdominal organs results. The stomach, intestine and liver suffer from venous engorgement. Gastric and intestinal motor and secretory functions become perverted and the adequate nutrition of the patient is with difficulty maintained.

So far as the stomach is concerned, the symptoms are similar to those of gastritis. Epigastric fulness, dull pain, anorexia with sickness and vomiting may all be present. Large quantities of mucus may be vomited along with undigested food material, and sometimes slight hæmorrhage occurs.

A vicious circle is set up and the disorder of the stomach, produced by the cardiac lesion, reacts on the function of the heart and aggravates the primary disease.

Treatment of cardiac dyspepsia is essentially that of the heart lesion. Usually large doses of cardiac tonics are indicated, but as these are almost all intrinsically irritant to the mucosa it is inadvisable to administer them by the mouth. They should be given hypodermically or intra-venously.

The gastric condition needs no less attention. Generally speaking the treatment should be on the same lines as that employed for gastritis. The venous congestion is often greatly relieved by free bleeding and the occasional use of a blue pill followed by a saline purgative. These remedies have also a favourable action on the heart itself.

7. Chronic Nephritis.

The dyspepsia of chronic renal disease is due to toxæmia when the stomach becomes the seat of gastritis due to the action of the toxins which it is attempting to eliminate.

The gastric disorder may be slight or severe according to the degree of toxæmia present.

In mild chronic uræmia flatulence, gastric uneasiness, loss of appetite, occasional nausea and vomiting, with irregular action of the bowels, may all be present.

When the uræmia is acute and toxæmia marked the symptoms are those of severe and acute gastritis. There are usually pronounced epigastric pain and tenderness, persistent vomiting and often violent and intractable diarrhœa. The vomitus is mucoid, is frequently blood-stained or 'coffee grounds' in appearance from hæmorrhage from superficial erosions of the gastric mucosa.

In addition to these symptoms of digestive derangement, there are more or less pronounced constitutional disturbances. Of these severe frontal headache, diplopia, somnolence and muscular twitchings are the most usual.

The prognosis depends on the renal condition, but is almost always grave.

Treatment is that of the renal trouble. It should aim at aiding elimination from all the channels available. Hot wet packs, diuretics, purgatives if diarrhœa be absent, pilocarpine and intravenous saline injections may all be employed in the treatment of this condition.

8. Diabetes Mellitus.

In the earlier stages of diabetes stomach symptoms are absent. Indeed, the gastric functions may be exalted and the increased appetite is associated with hyper-motility and hyper-secretion.

But, as in the case of chronic nephritis, when toxæmia becomes a feature of the disease, symptoms of chronic gastritis soon develop. A vicious circle is thus set up, for the failure of gastric function increases the severity of the diabetic symptoms.

Both the secretory and motor functions become defective. We may get marked atony, hypochlorhydria or achylia (*see* achylia gastrica).

When acidosis becomes well-marked gastric pain and tenderness, with nausea and vomiting, are common. The vomitus is often highly charged with acetone and has its characteristic odour.

Similar constitutional disturbances to those associated with uræmia characterise diabetes if the acidosis be pronounced.

The prognosis is grave, and treatment is directed to promote elimination and neutralisation of the acidosis. Gastric lavage often stops the vomiting, and large doses of sodium bicarbonate should be given rectally or intra-venously.

9. The Dyspepsia of Anæmia.

(*a*) **Chlorosis.**—In chlorosis dyspeptic symptoms are often pronounced. Various disorders of gastric sensation, secretion and motility may be present. As a rule there is a hyperchlorhydria and symptoms are apt to come on one to two hours after food. The association of gastric ulcer with this affection has already been discussed (*see* page 199).

(*b*) **Pernicious Anæmia.**—In the early stages of the disease symptoms are often absent. In my experience achylia is invariably present in every case of pernicious anæmia and is one of the earliest signs of this disease. It is of the simple type, although it may give rise to anxiety as to the presence of malignant disease (*see* achylia).

There may be intermittent attacks of pain in the gastric region, and diarrhœa is not uncommon. Glossitis is sometimes seen, though it is, I think, not so common as the literature on the subject might lead one to expect. The buccal mucosa is often inflamed and there may be superficial ulceration. Pyorrhœa alveolaris is usually present, and the breath is often very offensive.

Hæmorrhage from the stomach, as from the other mucous tracts, is not infrequent.

Treatment of the dyspepsia of pernicious anæmia is that of the blood disease.

A liberal diet should be given with as much blood-forming material as possible. Raw meat pulp and raw meat juice are useful, and red bone marrow with malt is an excellent supplement to the diet. Oral antiseptics are indicated and hydrochloric acid and arsenic should be given internally. The intramuscular injection of galyl I have found very effective in promoting rapid improvement in the blood picture and general condition.

CHAPTER XXVI.

VOMITING AS A SYMPTOM.

In many instances it is very difficult to allocate this symptom to its proper cause. This will be readily understood when it is considered how diverse are the conditions which may produce it.

Nausea, with salivation, usually precedes vomiting, but there is a type in which it is absent. The nausea may be regarded as nature's preparation for vomiting, for it probably causes, along with general muscular relaxation, relaxation of the sphincters. The act of vomiting involves both a muscular and nervous mechanism. The vomiting centre is situated in the medulla close to the respiratory centre, and stimulation of the one is apt to cause contiguous irritation of the other. It is thus that the respiratory rate may be increased during nausea, and violent coughing frequently ends in vomiting, as is well exemplified in pertussis.

The vomiting centre may be stimulated directly by toxins in the blood (*e.g.*, apomorphine) or reflexly by many forms of peripheral irritation. It is this multiplicity of conditions, which may excite it, that makes vomiting as a symptom of no great diagnostic value. A study of the entire case, with its history, and the correlated symptoms will, as a rule, enable the observer to make a correct diagnosis.

In certain forms of œsophageal obstruction food may be regurgitated from the dilated gullet in such quantities that it may erroneously be regarded as true vomitus. Chemical testing will usually prevent this error.

Persistent vomiting must always be treated seriously, and never put down to functional or reflex disorder till all other causes can be excluded.

In all cases of intestinal obstruction vomiting, sooner or later, becomes an important symptom. It is a feature of most cases of acute appendicitis and peritonitis.

Bilious vomiting.—Quite commonly when vomiting is associated with much straining and retching, as in sea-sickness and migraine, the vomitus is bile-stained or even pure bile. But in such cases the bilious vomiting is not persistent. Small quantities of bile seen in the last of the vomit are not of any significance, but are merely due to regurgitation into the stomach.

In an entirely different category is the persistent bilious vomiting of obstruction of the lower part of the duodenum below the entrance of the hepatic duct into the bowel. When bilious vomiting is persistent and large in amount interference with its passage down the gut should be suspected. Evidence of obstruction may be present in other signs and symptoms.

Nervous vomiting.—A type of this is sometimes met with in hysterical women. It is intermittent in character, is related to ingestion, and is usually small in amount. There is singularly little or no emaciation, and no other signs of digestive derangement. It occurs under conditions which usually produce or accompany other hysterical manifestations.

Vomiting may occur in certain subjects from psychological disturbances. Sudden shock, the abrupt announcement of bad news, fright, a gruesome tale or sight, suffice to induce vomiting.

Periodical nervous vomiting is a less common affection. The attacks are apt to come on periodically and suddenly when the patient appears in good health, and when no obvious cause for the disturbance is ascertainable. There is often complete intolerance of all fluid and solid alimentation for days on end, causing much prostration and subsequent debility. Auto-intoxication, with acetonæmia, is apt to supervene. The attack ends as abruptly as it began. Periodic nervous vomiting being a rare affection should not be diagnosed till other possible causes have been considered. Thus the nervous vomiting of locomotor may be overlooked. Other evidences of tabes should be carefully sought for, and a Wassermann reaction of the blood and cerebro-spinal fluid carried out.

A more severe form of vomiting associated with recognised locomotor-ataxy is that of the *gastric crisis*. In this type the vomiting is generally associated with epigastric pain, which radiates in all directions, and by mental distress. It persists for a variable time, and often ends suddenly, leaving the patient very exhausted.

Various *cerebral lesions* are associated with vomiting of a more or less characteristic type. Such emesis is usually easy, painless, explosive, and unassociated with nausea, and is not necessarily related to the act of ingestion. It is well exemplified in cerebral tumours and meningitis, especially at the base.

Violent vomiting commonly occurs in *Menière's Disease* (labyrinthine vertigo). Sea-sickness has been ascribed to many causes, but probably it too has a cerebral origin.

The vomiting of pregnancy.—Not less than 80 per cent. of pregnant women are afflicted with dyspepsia during the period of gestation. The symptoms which accompany it are so distressing, and at times even so dangerous, that it requires special mention.

The chief feature of the condition is morning sickness and vomiting. These are probably only symptoms of a chronic gastritis due to some toxæmia associated with pregnancy. It is impossible to regard it as wholly due to reflex irritation, as is commonly done, for it does not conform in type to the true 'nervous' or 'reflex' sickness.

The dyspepsia of pregnancy takes many forms in different women. Perhaps the most prominent symptom is that of morning sickness. The patient is usually attacked with violent retching on getting out of bed. But sickness is not infrequently intimately associated with the ingestion of food, which prevents the pregnant woman from finishing her meal. As a rule vomiting is confined to the early months of pregnancy, but it may be much more intractable and last throughout the entire period. It then causes alarming failure of nutrition and can be so severe that premature labour may have to be induced to save the patient's life.

In organic disease of the stomach vomiting is usually definitely related to the act of eating. The special features of such vomiting have been dealt with under the different diseases of the stomach, more particularly in discussing cancer and peptic ulcer.

If we can exclude a lesion of the stomach and the digestive tract it is not always easy to say whether the vomiting is symptomatic or nervous. Symptomatic vomiting occurs in many conditions, as in Bright's disease and uræmia. Nervous vomiting may be hysterical, periodic, or psychic, and we have vomiting occurring frequently and persistently from many peripheral and central reflex irritations as, for example, from biliary and renal colic.

Treatment of Vomiting.—It should be borne in mind that in one sense all vomiting is due to irritation or excitation of the vomiting centre, and that whatever treatment be adopted one part of it should be to diminish the receptivity of that centre to excitation locally or reflexly.

The other principle of treatment is to obtain gastric rest.

The exact measures we may recommend for the relief of vomiting will obviously depend on the diagnosis of its cause.

If due to an organic lesion of the stomach—peptic ulcer, cancer, or gastritis—the vomiting associated with those conditions will best be dealt with by the general management of these diseases.

Increased elimination through all the excretory channels is indicated in toxic vomiting. Thus in the vomiting of acute alcoholic gastritis and in that of uræmia purgation, diuresis and diaphoresis are all indicated. At the same time the stomach should be kept at rest by fasting and the administration of sedatives.

Certain measures may be adopted with advantage, irrespective of the exact cause of the vomiting. Thus rest in bed is essential, and usually the patient should be kept fasting.

Sinapisms and warm compresses applied to the epigastrium are almost always grateful to the patient. Chips of ice, in certain cases, serve to allay gastric irritation, and very hot water, sipped slowly, may have a similar effect.

If irritant or toxic material be present in the stomach an emetic is usually a most efficacious remedy or gastric lavage may be still better.

The medicinal treatment of vomiting which we may adopt will depend on the view we take of its causation. The following formulæ contain remedies which have a sedative action on the gastric mucosa or on the vomiting centre :—

℞	Cerii Oxalatis	℥i.
	Bismuthi Oxidi	℥ii.
	Mucilaginis	℥iss.
	Spiritūs Menthæ Piperitæ	℥ss.
	Aquæ Chloroformi....	ad ℨvi.

M.

Sig. :—℥iv, in water, as may be required.

℞ Tincturæ Belladonnæ
Nepenthe .... āā ℥i.
Sodii Citratis .... ℥ii.
Spiritus Chloroformi .... ℥i.
Aquæ Menthæ Piperitæ .... ad ℥vi.
M.

Sig. :—℥iv, in water, as may be required.

℞ Potassii Bromidi .... ℥ii.
Chloral Hydratis .... ℥i.
Syrupi Codeinæ .... ℥vi.
Aquæ Anethi .... ad ℥vi.
M.

Sig. :—℥iv, in water, as may be required.

℞ Chloretone .... ℥i.
Spiritus Menthæ Piperitæ .... ℥ss.
Bismuthi Hydratis .... ad ℥vi.
M.

Sig. :—℥ii, in water, as may be required.

℞ Acidi Hydrochlorici diluti .... ℥iv.
Glycerini Pepsini .... ℥ii.
Syrupi Limonis .... ℥i.
Aquæ .... ad ℥vi.
M.

Sig. :—℥ii, in water, as directed.

(Useful in the vomiting of pregnancy.)

Sea-sickness is frequently relieved by the hypodermic injection of atropine or pituitrin. The drugs should be used before going on board and at intervals during the first few days of the voyage. I have recently seen atropine so used most efficacious in preventing sea-sickness in those who had previously been very bad sailors.

CHAPTER XXVII.

CONSTIPATION.

Constipation may be acute or chronic. The former is passing and not of serious importance unless dependent on organic disease, such as intestinal obstruction.

Chronic Constipation.—The causes of habitual constipation are so varied and numerous that it is impossible even to enumerate them all in a work chiefly concerned with gastric disorders.

There are, however, broadly speaking, four operative causes :—

1. Diminished peristalsis—*e.g.*, atony ; enteroptosis.
2. Diminished secretion.
3. A combined diminution of motor and secretory function.
4. Excessive absorption from the bowel to compensate for excessive functional activity of skin and kidney, as for example in excessive sweating and diabetes.

Many kinds of contributary factors may be added to these, as improper diet, want of exercise, and congenital tendency.

Habitual constipation may be classified into two types—atonic and spastic.

Atonic Constipation.—This is the commonest type met with. To stimulate its contraction the bowel normally requires a dietary which leaves an adequate residuum. Foods which are almost entirely soluble and leave little residue, such as eggs and meat, have no stimulant action on the bowel, and are apt when they form too exclusive a part of the diet to induce constipation.

Want of exercise and fresh air also beget intestinal atony. It is for this reason that habitual constipation is so much more common among an urban than a rural population.

Atonic constipation is an accompaniment of gastroptosis and enteroptosis and of relaxation of the abdominal muscles.

Spastic Constipation.—This is chiefly due to over-tonicity of the large intestine. It is not usual that the spastic condition affects the entire colon but is commonly found in parts where the musculature

is best developed. The lumen of the gut is contracted and may retain the intestinal contents or the spastic condition may be sufficiently developed almost to amount to an actual obstruction. In such cases, on palpation of the left iliac fossa, the sigmoid flexure may be felt contracted and corded as if containing scybalæ in its interior.

A more diffuse enterospasm is met with in certain lesions of the central nervous system. This accounts for the scaphoid abdomen and the obstinate constipation associated with basal meningitis and diseases of the pons and medulla.

The toxic influence of lead also causes serious enterospasm, colic and obstinate constipation.

Spastic constipation is most commonly found in the young and neurotic.

Symptoms of Constipation.—By exception some people seem to suffer no ill effects from habitual constipation. On the other hand the vast majority are adversely affected and suffer symptoms of varying intensity and infinite variety. These are constitutional and local.

The constitutional disturbances may chiefly be ascribed to auto-intoxication. Flatulence, nausea, vomiting, vertigo, anorexia and headache are commonly experienced. The tongue is frequently dirty, the complexion muddy and sallow, and there is often a degree of anæmia. The breath is often very offensive. In some individuals there may be cardio-vascular symptoms, such as palpitation, arhythmia and vaso-motor disorders. Sometimes nervous symptoms are associated with habitual constipation such as passing attacks of aphasia, migraine and chronic insomnia. Piles are apt to develop and, in the elderly, fæcal accumulations may form in the rectum.

Locally abdominal discomfort is usually experienced. Colic, distension, dragging and dull pains or aching in the left hypochrondriac region are of common occurrence.

The irritation set up from long-continued retention of fæces may result in a catarrhal condition and induce a diarrhœa which is shortly replaced by the habitual constipation.

Treatment.—Constipation has such wide-spread pathogenic influences that no functional derangement requires more attention and treatment. It is far too often neglected and advice only sought when some of its complications overtake the patient.

Acute constipation may usually be overcome by the administration of some drastic purgatives to clear the bowel and by subsequent regulation of the diet. For this purpose one or two drops of croton oil or four grains of compound powder of elaterinum may serve well. If something less drastic is considered sufficient this pill may be prescribed :—

℞ Aloini .... gr. 1.
Extracti Colocynthidis .... gr. i.
Resinæ Podophylli .... gr. ½.
Extracti Hyoscyami .... gr. ¼
Olei Caryophylli .... mi.
Ft. Pil. Mitte tales 12. M.

Sig. :—One pill at bedtime, or as may be required.

Habitual Constipation.—When possible no treatment should be prescribed that will needlessly interfere with the daily occupations and necessitate abandonment of business.

(1) *Relief of the bowels* ought to be sought at a fixed hour till a regular habit has been established. There is no doubt that intestinal evacuation is largely a matter of habit. Nature's call for relief, by setting up peristalsis in the colon, is a periodic one, and many people find it difficult to pass the habitual hour. Indeed chronic constipation is sometimes begotten by frequently ignoring or restraining the desire to defæcate.

(2) *Good hygiene*, in its widest sense, should be insisted on in all cases where it is defective. Fresh air, sanitary surroundings, adequate rest and exercise must not be overlooked.

Exercise is most important. A too sedentary occupation is one of the chief pathogenic influences in producing chronic constipation. There are too many facilities for transit in these days, and we run the risk of losing the use of our legs and acquiring habitual constipation. Every sufferer should be counselled to walk to and from his business, or if this is impracticable a daily walk should be insisted on. For those who can afford it no exercise is more beneficial than horse riding, and golf can also be recommended for those who have some leisure.

Physical culture, in addition to out-of-door exercise, can also be highly recommended as a powerful remedial agent in the treatment of habitual constipation. It may in some instances require to be substituted for out-door exercise.

The exercises which I have found most useful are those of Müller.* They have the advantage of requiring no apparatus for their performance.

For business men who have no leisure in the morning they are best undertaken before going to bed. The bath, which is advised in the middle of the exercises, may be dispensed with when it is contraindicated or cannot conveniently be obtained. But the exercises, massage, and deep-breathing should never be omitted. If this scheme of physical culture is not adopted the physician may recommend special exercises which will develop the abdominal muscles and act on the hollow viscera. Not infrequently an enthusiastic beginning is made, but anon, because no immediate relief follows, the exercises are abandoned. It is therefore sometimes advisable to advise young patients to join a class of physical culture.

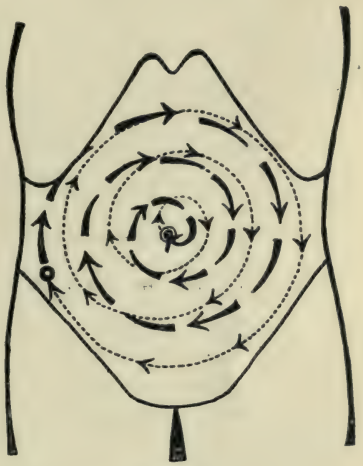


Fig. 31.

(3) Massage. For all those who are unable to take exercise and physical culture, general and abdominal massage is specially useful.

Auto-abdominal massage, for fifteen minutes every morning before rising, is an excellent mode of stimulating the bowel. With the knees flexed and the patient lying on his back deep auto-massage can be carried out fairly efficiently. The physician can give the few simple instructions which are required. The colon should be massaged, by means of the clenched fist, in the direction of its peristalsis from the caput to the hepatic flexure, across transverse colon to the splenic flexure and down descending colon to sigmoid flexure. On the completion of every six circular movements, from right to left, the circle should be gradually reduced till the umbilicus is reached and then gradually enlarged till the periphery is again reached. In this way both the large and the small intestines are influenced.

* "My System," by Lt. J. P. Müller, can be obtained at booksellers. There are different schemes for men and women.

(4) Dietetic Treatment.—There seems little doubt that a too great refinement in our diet has bred a constipated generation. The over-refinement of our flour and our bread, due to the perfection of milling processes, is doubtless an important factor in predisposing to constipation. In the days of hand mills, when cereals were not reduced to impalpable powders, constipation was far less common. The form of diet must, therefore, be carefully prescribed.

If there be no serious derangement of the stomach the following articles of food, leaving a large residuum, may be recommended: Porridge and oatcakes may be advised, while whole-wheat meal bread may be substituted for the fine white loaf. Almost all kinds of fruit are specially indicated, and ripe raw fruits are to be preferred to those cooked. Vegetables, raw and cooked, should form a liberal part of the diet. Salads are particularly useful, and vegetable soups are to be recommended.

Treacle and all cakes made with it may also be taken freely. The variety known as Fowler's West Indian treacle is very palatable, and may be used with bread and butter as a substitute for preserves.

Cold water or certain other fluids should be freely taken between meals. Constipation is certainly diminished by keeping the intestinal tract well supplied with liquids. A plentiful supply of water also helps to wash out the toxins and prevents auto-intoxication.

(5) Medicinal Treatment.—The fewer drastic purgatives resorted to the better for the patient. The bowel ceases to react even to violent purgatives if it is habitually over-stimulated.

For the treatment of constipation of Plumbism two drops of croton oil, or four grains of compound powder of elaterinum, may be repeated for the first three days of treatment, and then followed by a saline sulphate to maintain evacuation. For example:—

℞	Magnesii Sulphatis	℥vi.
	Sodii Sulphatis	℥vi.
	Syrupi Aurantii	℥iss.
	Extracti Glycyrrhizæ Liquidi	℥ii.
	Infusi Rosæ Acidi .... ad	℥vi.
		S. et M.

Sig.:—℥i, in half-a-pint of water, to be taken twice or thrice daily.

For the treatment of all other types of acute constipation any of the active purgatives may be used and there is nothing better than a large dose of castor oil.

For habitual constipation hygienic, cultural, and dietetic treatments may not alone suffice without the co-operation of laxatives. These may therefore be used for a time, but the dose should be steadily reduced till all medicinal treatment is entirely withdrawn.

The physician may find some of the following formulæ useful. One or other of these may be indicated for special reasons or to combat some obtrusive or troublesome symptoms.

(1) Either of these formulæ may be found useful if anæmia complicates the case.

℞ Ferri Sulphatis Exsiccati .... gr. ½.
Ferri Arsenatis .... gr. $\frac{1}{36}$
Aloini .... gr. ½.
Extracti Rhei .... gr. ½.
M.

Ft. Pil. Mitte tales 60.
Sig. :—One pill, thrice daily, after food.

℞ Ferri Redacti .... gr. ii.
Ferri Arsenatis .... gr. $\frac{1}{36}$
Thyroidei Sicci .... gr. $\frac{1}{12}$
Extracti Euonymi Sicci .... gr. ½.
Extracti Cascaræ Sicci .... gr. ½.
M.

Ft. Pil. Mitte tales 60.
Sig. :—One pill, thrice daily, after food.

(2) For habitual use to relieve the common digestive derangements of chronic constipation :—

℞ Extracti Sennæ Fructus Liquidi
Cascaræ Evacuant .... āā ℥vi.
Tincturæ Podiphylli .... ℥iss.
Extracti Glycyrrhizæ Liquidi .... ℥ii.
Decocti Aloes Compositi .... ad ℥vi.
M.

Sig. :—℥ii, in water, two or three times daily after food.

℞ Extracti Rhei
Extracti Cascaræ Sicci
Extracti Hyoscyami
Aloini .... āā gr. ½.
Ft. Pil. Mitte tales 24. M.
Sig. :—One pill, thrice daily, after food.

℞ Magnesii Sulphatis
Extract Sennæ Fructūs Liquidi .... āā ℥vi.
Tincturæ Rhei .... ℥iv.
Syrupi Zingiberis .... ℥i.
Aquæ Menthæ Piperitæ .... ad ℥vi.
Sig. :—℥iv, in water, thrice daily. M.

℞ Sodii Bicarbonatis
Sodii Sulphatis Exsiccati .... āā ℥i.
Sodii Phosphatis Exsiccati .... ℥vi.
Phenolphthaleine .... gr. 24.
M.
Sig. :—℥i—ii, in a tumblerful of hot water, one hour before breakfast.
(On the addition of water a pink solution will result owing to the action of the alkali on the phenolphthaleine.)

℞ Olei Ricini
Glycerini .... āā ℥iii.
M.
Sig. :—℥i—ii, to be taken in the early morning, as directed.

Liquid paraffin, olive oil, cotton-seed oil, agar-agar, and glycerin suppositories may all be used when it is necessary to employ aperients regularly.

(3) Should ' biliousness ' and a dirty tongue be specially marked these formulæ may prove suitable :—

℞ Pilulæ Hydrargyri.... .... gr. ½.
Resinæ Podophylli .... gr. $\frac{1}{6}$
Aloini .... gr. ¼.
Pilulæ Colocynthidis Compositæ.... .... gr. ii.
M.
Sig. :—One pill, to be taken every night at bedtime for a week.

℞ Extracti Euonymi Sicci .... gr. i.
Iridini .... gr. $\frac{1}{8}$.
Hydrargyri Subchloridi .... gr. $\frac{1}{2}$.
Aloini .... gr. $\frac{1}{2}$.
Extracti Taraxaci .... gr. ii.
Ft. Pil. Mitte tales 24. M.
Sig. :—One pill, morning and evening.

(4) If flatulence be specially troublesome :—
℞ Salol .... gr. ii.
Gingerine .... gr. $\frac{1}{4}$.
Olei Carophylli .... m. i.
Pilulæ Aloes et Asafœtidæ .... gr. ii.
Ft. Pil. Mitte tales 24. M.
Sig. :—One pill after each meal.

(5) Should constipation be complicated by piles this formula will be found useful :—
℞ Confectionis Sulphuris
Confectionis Piperis
Confectionis Sennæ .... āā ℥i.
Sig. :—℥i, at bedtime. M.

(6) If headache be severe a sedative may be combined with the aperient :—
℞ Acetanelidi
Caffeinæ .... āā gr. i.
Resinæ Podophylli .... gr. $\frac{1}{24}$
Pilulæ Rhei Compositæ .... gr. ii.
Ft. Pil. Mitte tales 24. M.
Sig. :—One pill after each meal.

(7) If arhythmia and vaso-motor disturbances complicate the case this formula may be useful :—
℞ Extracti Strophanthi Sicci .... gr. $\frac{1}{3}$.
Liquoris Trinitrini.... .... m. i.
Strychninæ.... .... gr. $\frac{1}{36}$
Extracti Belladonnæ .... gr. $\frac{1}{4}$.
Aloini .... gr. $\frac{1}{2}$.
Ft. Pil. Mitte tales 36. M.
Sig. :—One pill thrice daily, after food.

(8) When enteroptosis is predominant better tonus and peristalsis may be promoted by this pill :—

℞	Strychninæ	gr. $\frac{1}{30}$
	Physostigminæ Sulphatis	gr. $\frac{1}{90}$
	Extracti Belladonnæ	
	Extracti Ergotæ	āā gr. ¼.
	Olei Menthæ Piperitæ	m. i.
	Aloini	gr. ¼.
	Ft. Pil. Mitte tales 24.	M.

Sig. :—One pill, thrice daily, after food.

(9) When aperients by the mouth fail to give relief we must resort to mechanical measures. In certain cases all we may try is of no avail. I frequently see people who adopt the regime and diet set out in this chapter and who can swallow incredible quantities of the strongest purgatives without any appreciable relief. When such cases are met with it is far safer to rely on rectal douches, administered as high as possible, for the relief of the bowels. A strong purgative may be used twice a week to aid the descent of the fæces.

As an injection magnesium sulphate (℥iv to the one pint) dissolved in warm water, or in normal saline solution, answers well. If this form of enema fails a more purgative one of soap, oil, and water may be introduced, or one of glycerin and hot water (℥ii to ℥iv). If the glycerin is dissolved in normal saline it is much less irritant.

Cotton-seed oil (℥iii-vi) injected into the rectum at bedtime and left in over night usually results in defæcation in the morning, or it makes the action of enemata much more certain.

Various other methods of dealing with chronic constipation may be considered.

If the patient can afford it treatment at a spa may be advised. Harrogate, Bath, Strathpeffer, Droitwich at home ; and Spa, Carlsbad, Vichy and many other places abroad, offer certain therapeutic influences which are valuable in the treatment of chronic constipation.

Electro-therapy may also be utilized. High frequency current and Bergonié's treatment act as stimulants of intestinal peristalsis.

Hydro-therapy is also worth trying, and I have seen several cases which resisted all other forms of treatment yield to hypnotic suggestion.

Within recent years surgical interference for intractable constipation has been advocated, especially by Lane. It requires some courage in the physician to enter a protest against the resection of the colon for constipation, which is apparently otherwise incurable. Theoretically a short-circuit ought to ensure relief, by bringing the fluid contents of the ascending colon to the sensitive sigmoid flexure, but in fact it often does not do so. I have recently been dealing with a number of patients on whom resection of the colon has been performed who remain as constipated as before the operation. If there be almost complete atony of the whole tract I do not see how resection of the colon should be expected to yield good results. It is thus reasonable to be somewhat sceptical of the claims that have been advanced for this procedure as a cure for chronic constipation.

To summarise, the following is an outline of the daily programme which may be advised for the treatment of chronic constipation :—

7-30 a.m.—A glass of cold or hot water should be sipped slowly. If the physician thinks it advisable, to this may be added some magnesium sulphate, sodium sulphate, sodium phosphate, sodium bicarbonate, or a little cascara evacuant. Or a few broken-up senna pods may be placed in a tumblerful of boiling water and allowed to infuse over night. Next morning the cold infusion may be slowly sipped one hour before breakfast.

8 a.m.—Abdominal auto-massage should be practised for ten minutes, and when the patient rises exercises may follow. Cold or sponge bath (?).

8-30 a.m.—*Breakfast.* This should consist of porridge, brown bread (whole wheat meal), oatcake, ginger-bread, treacle. Fish, bacon, eggs, etc. Tea or café au lait and fruit (figs, prunes, apples, bananas, etc.).

11 a.m.—Glass (12ozs.) of cold or hot water, or Vichy water, etc., to be sipped slowly.

1 p.m.—*Lunch or Dinner.* Vegetable soup. Some flesh (meat, fish, chicken, game, etc.). Vegetables as separate course. Fruit. Brown bread and cheese. (No milk puddings.)

4 p.m.—Hot or cold water, or a large cup of China tea with sugar and milk.

7 p.m.—*Dinner or Supper.* Vegetable soup. If red flesh was taken at 1 p.m. white flesh should be eaten at 7 p.m. Potatoes. Vegetables as separate course. Fruit. Brown bread. (No milk puddings or coffee.)

9 p.m.—Müller's exercises.

Observations on Above Instructions.

(1) Unless specially contra-indicated the patient should take a cold bath or cold sponging before breakfast.

(2) A daily walk of at least a couple of miles should be advised.

(3) The patient should be warmly clothed. The feet should be kept dry and warm.

(4) From the outset of treatment the patient should begin to educate the bowel to periodic action by going to the W.C. daily at a fixed hour even though there be no special 'call' to do so. He may exert gentle pressure but never strain, and rather than fail to accomplish evacuation, he should use a child's-size glycerin suppository.

The best time to cultivate the habit of defæcation is after breakfast. But this may not be suitable for those who have no time to spare in the morning, nor is it advisable for those suffering from piles. Defæcation aggravates hæmorrhoids by increasing local congestion. If the bowels are relieved at night and immediately thereafter the patient goes to bed, local discomfort and congestion are mitigated. In addition to the remedies which may be used internally local treatment for piles should never be neglected. Immediately after defæcation the anal orifice should be well douched or sponged with cold water. The remnants of fæcal matter around the orifice are thus removed efficiently and irritation prevented. If this procedure is carried out persistently and regularly it will be found a potent prophylactic against piles and pruritus ani.

If piles exist, after the anal orifice is thoroughly cleansed, these ointments, put up in flexible tubes with rectal nozzle, are very palliative. If the tube is placed before the fire for a few minutes before being used the contents are much more easily expressed :—

℞ Eucainæ Lactatis	....	4%
Solutionis Adrenalini	....	℥iii.
Olei Rusci		
Olei Cadini	....	āā m20.
Olei Olivæ	....	℥ss.
Adipis Lanæ	....	ad ℥iss.
		M.

Ft. Ung. (Put into flexible tube with rectal nozzle.)

Sig. :—Use as directed.

℞ Adipis Lanæ .... ℥ii.
Orthoform 2%
Extracti Conii .... ℥ii.
Solutionis Adrenalini .... ℥ii.
Unguenti Gallæ c̄ Opiō .... ad ℥i.
M.

Ft. Ung. (Put into tube.)
Sig. :—Apply as directed.

℞ Extracti Hamameledis .... ℥ii.
Eucainæ Lactatis 4%
Solutionis Adrenalini .... ℥ii.
Adipis Lanæ .... ℥iii.
Calaminæ .... ℥i.
Unguenti Gallæ c̄ Opiō .... ad ℥ii.
M.

Ft. Ung. (Put into tube.)
Sig. :—Apply as directed.

(5) If there be no signs of hyperchlorhydria moderate smoking may be encouraged. Often a pipe or cigar immediately after breakfast excites the necessary peristalsis which induces the bowels to move.

CHAPTER XXVIII.

DIARRHŒA.

Like constipation, diarrhœa may be acute or chronic. The very opposite conditions to those which may be responsible for constipation are frequently operative in producing diarrhœa.

Intestinal hyper-motility or hyper-secretion, or both in combination, or a failure of intestinal absorption may all be pathogenic factors in diarrhœa. The hyper-motility may be confined to the small or large intestine, or it may extend throughout the entire tract.

The rectum is intolerant of fluid even in health, but when it is inflamed it resents its contents and diarrhœa ensues. When chyme is hurried down to the lower bowel without sufficient time for absorption to take place it may result in diarrhœa.

The stools may be few or many, and vary greatly in consistency according to the amount of water they contain. They may be pultaceous or absolutely fluid.

The appearance of the stools is of importance for diagnosis. If the food material is badly digested and recognisable we may conclude that the small intestine is at fault (lienteric diarrhœa).

In the normal fæces mucus is present in such small amount as hardly to be recognisable macroscopically. When the very constipated motion lies in contact with the mucous membrane of the colon for too long a period it locally excites a catarrh which coats the surface of the hardened fæces with mucus, so in association with scybalæ it is, when small in amount, of no importance.

Mucus from the lower part of the colon is not bile-stained, while that from the upper reaches and from the small intestine usually is.

The presence of excessive quantities of mucus is indicative of enteritis or colitis. It may be like starch mucilage or in the form of casts or flattened shreds (membranous colitis). Mucus streaked with blood is commonly passed in malignant disease of the rectum.

Diarrhœa may be Functional or Organic.

(1) Functional diarrhœa may arise from a great variety of causes. It may be due to the local irritation of unsuitable or excessive food which has been ingested. Certain gastric conditions, even though there be no error in the quality or quantity of the food ingested, may be provocative of diarrhœa. Thus it may be a troublesome sequel to gastro-enterostomy due to the precipitate emptying by the stomach of its ill-prepared chyme into the bowel. The diarrhœa of achylia gastrica has probably a similar origin.

Diarrhœa may be eliminative in character when the intestinal mucosa attempts to throw out certain toxic substances which are circulating in the blood. A good example of this is seen in diarrhœa of uræmia. Or diarrhœa may result from constipation—an attempt on nature's part to eliminate from the bowel fæcal accumulations.

Another form of diarrhœa is purely *nervous* in origin (diarrhœa nervosa). Sudden emotion, anxiety, grief, etc., may in neurotic subjects induce an attack of diarrhœa. We have frequently examples of this variety in hysteria and neurasthenic conditions. The diarrhœa of ingestion belongs to the same category. The contact of food with the hyper-excitable gut evokes an attack of looseness of the bowels shortly after food is taken.

Severe paroxysmal diarrhœa may occur in acute and advanced cases of Grave's disease.

A large number of infective diseases of the bowel are also associated with diarrhœa. Thus it is seen in cholera, enteric fever, sprue, general tuberculosis, pernicious anæmia, Addison's disease, and acute infective gastro-enteritis. The summer diarrhœa of children occurring principally in bottle-fed infants is associated with very alarming symptoms, and unless controlled may rapidly prove fatal.

Diarrhœa may also be caused by almost any of the chemical corrosive or irritant poisons—*e.g.*, arsenic, antimony, and alkaloidal poisons—*e.g.*, ptomaines.

Habitual diarrhœa and retching in the morning, when the patient gets out of bed, is very suggestive of chronic alcoholism. It is associated with a catarrhal condition of the gastro-intestinal mucosa.

(2) The diarrhœa due to organic lesions, such as ulceration of the bowel, is more intractable and persistent than that which is associated with functional derangements.

In dilatation of the heart, from failure of compensation, the sudden onset of diarrhœa, with evacuation of blood-stained mucoid

stools, severe pain and abdominal distension, is very suggestive of intestinal infraction.

While constipation is more usually associated with *appendicitis*, it should be kept in mind that diarrhœa is not infrequently the initial symptom of this disease.

Treatment of Diarrhœa.—As peristalsis is promoted by the movements of the body and the erect posture, one of the most important indications for the treatment of this affection is rest in bed. Tissue waste is thus also diminished.

Next in importance is an appropriate dietary. The articles we select should be nutritious, bland, and leave little detritus. Egg albumin, boiled milk and lime water, curds, butter-milk, arrowroot gruel, jellies, and small quantities of port wine or brandy may be selected as suitable.

While rest and diet are essential for all forms of diarrhœa, the exact causation of the ailment will largely determine our choice of medicinal agent.

For ordinary acute diarrhœa due to chill or ingestion of unsuitable or impure food a preliminary dose of castor oil, with a few drops of laudanum, followed by astringents, often answers well. When the castor oil has been evacuated the quickest and most certain way of arresting diarrhœa is to give a starch and opium enema (20 to 30 drops of laudanum to ℥iii of starch mucilage).

Certain medicinal agents may also be orally administered :—

℞ Bismuthi Salicylatis .... ℥ii.
Tincturæ Camphoræ Compositæ .... ℥vi.
Tincturæ Cardamomi Compositæ .... ℥iv.
Misturæ Cretæ .... ad ℨvi.
M.

Sig. :—℥iv, in water, after each loose stool.

℞ Salol
Bismuthi Subnitratis
Pulveris Cretæ Aromatici c̄ Opiō āā gr. xv.
M.

Ft. Pulv. Mitte tales 24.

Sig. :—One powder, after each loose stool.

℞ Tincturæ Rhei
Tincturæ Kino .... āā ℥iv.
Tincturæ Chloroformi et Morphinæ Co ... ℥i.
Aquæ Cinnamomi .... ad ℥vi.
M.

Sig. :—℥iv, in water, as may be required.

℞ Acidi Sulphurici diluti .... ℥ii.
Nepenthe .... ℥i.
Tincturæ Ferri Perchloridi .... ℥iss.
Glycerini Pepsini .... ℥ii.
Syrupi Zingiberis .... ℥i.
Aquæ Chloroformi.... .... ad ℥vi.
M.

Sig. :—℥iv, in water, as may be directed.

For the *diarrhœa of achylia gastrica* and that of *gastro-enterostomy* :—

℞ Acidi Hydrochlorici diluti .... ℥vi.
Glycerini Pepsini
Syrupi Codeinæ .... āā ℥iss.
Syrupi Aurantii .... ℥vi.
Aquæ Cinnamomi .... ad ℥vi.
M.

Sig. :—℥ii, in water, t.i.d.p.c.

The *diarrhœa of uræmia* is very intractable. In renal disease it is not advisable to give large doses of opium. Narcotics must be used with caution. Diuretics and diaphoretics are indicated. Hot wet packs and vapour baths may also be helpful, and certain astringents exert a controlling influence :—

℞ Liquoris Ferri Penitratis
Potassii Citratis .... āā ℥iv.
Tincturæ Strophanthi .... ℥i.
Tincturæ Opii .... ℥ii.
Aquæ Cinnamomi .... ad ℥vi.
M.

Sig. :—℥ii, in water, thrice daily, or as may be required.

℞ Spiritūs Ætheris Nitrosi .... ℥vi.
Potassii Citratis
Potassii Acetatis .... āā ℥ii.
Syrupi Aurantii .... ℥i.
Liquoris Ammonii Acetatis .... ad ℥vi.
M.

Sig. :—℥iv, in water, as may be directed.

Nervous Diarrhœa.—Rest after food should be advised. The food should be of the astringent type—boiled milk, arrowroot gruel, egg-albumin, jellies, etc.

Sedatives and astringents are also employed with advantage.

For the special form, known as the *diarrhœa of ingestion*, small doses of arsenic act best :—

℞ Liquoris Arsenici Hydrochlorici .... m24.
Acidi Hydrochlorici diluti .... ℥iv.
Tincturæ Cinnamomi .... ℥iii.
Syrupi Limonis .... ℥i.
Infusi Rosæ Acidi .... ad ℥vi.
M.

Sig. :—℥ii, in water, thrice daily, after food.

For the other nervous or *reflex diarrhœas* these remedies are suggested :—

℞ Potassii Bromidi .... ℥iii.
Spiritus Menthæ Piperitæ .... ℥i.
Diamorphinæ Hydrochloridi .... gr. iss.
Syrupi Zingiberis .... ℥i.
Aquæ Chloroformi.... .... ad ℥vi.
M.

Sig. :—℥ii, in water, thrice daily, after food.

℞ Tincturæ Catechu
Tincturæ Camphoræ Compositæ.... āā ℥vi.
Syrupi Papaveris .... ℥i.
Liquoris Bismuthi .... ad ℥vi.
M.

Sig. :—℥ii, in water, thrice daily, after food.

Diarrhœa due to intestinal fermentation and putrefaction with foul-smelling stools is best treated by antiseptics and astringents :—

℞ Beta Naphthol .... gr. ii.
Thymol .... gr. ¼.
Morphinæ Hydrochloridi .... gr. ⅛.
Bismuthi Salicylatis .... gr. xv.
Ft. Cachet. Mitte tales 24. M.
Sig. :—One cachet every four to six hours.

℞ Bismuthi Carbonatis
Pulveris Kino Compositi
Salol .... āā gr. xv.
Ft. Pulv. Mitte tales 24. M.
Sig. :—One powder, twice daily.

℞ Creosoti Phenylpropionatis .... m v.
Thymol .... gr. ⅛.
Morphinæ Hydrochloridi .... gr. ⅛.
Olei Menthæ Piperitæ .... m i.
Ft. Capsul. Mitte tales 24. M.
Sig. :—One capsule, thrice daily, after food.

Creosote phenylpropionate passes through the stomach unchanged but is decomposed, with the liberation of creosote, by the alkaline intestinal secretions.

For *infantile diarrhœa*, if the case is seen early, a small purgative dose of castor oil, or of calomel, may be given. It is also very useful treatment to wash out the stomach with a weak solution of sodium bicarbonate or of borax. This sometimes arrests the vomiting. With the lavage may be combined flushing of the colon with warm normal saline solution.

After an interval medicinal treatment may be begun orally, and one of these powders may be found useful :—

℞ Pulveris Ipecacuanhæ Compositi .... gr. ⅛-¼.
Hydrargyri c̄ Cretâ .... gr. ¼.
Pulveris Rhei .... gr. ¼.
Sodii Bicarbonatis.... .... gr. i.
Ft. Pulv. Mitte tales 24. M.
Sig. :—One powder, mixed with a little glycerin, to be placed on the child's tongue as directed by the physician.

R Salol .... gr. ½-i.
Pulveris Ipecacuanhæ Compositi .... gr. ⅛-¼.
Bismuthi Carbonatis .... gr. ii.
M.

Ft. Pulv. Mitte tales 24.
Sig. :—To be used similarly to above.

The *diarrhœas associated with ulceration* of the bowel can only be palliated by opiates, diet, and astringents. Under certain circumstances, if the ulceration be limited and favourably situated, it may be dealt with surgically.

All forms of chronic diarrhœa which resist other treatments may be greatly relieved or cured by a prolonged ocean voyage.

GLOSSARY OF TESTS.

Laboratory Requirements.—The reagents and apparati usually found in the physician's test room will, with certain additions, suffice for the investigation of diseases of the stomach.

(*a*) Additional apparati :—

Assorted pipettes—plain and graduated.
Glass rods.
Small porcelain mortars and pestles.
Porcelain saucers (various sizes).
Strauss' separating funnel.
Schmidt's and Strassburger's fermentation tube.
Weiss' acidometer tube.
Buret.
Ewald's or Boas' stomach tubes (medium sizes).
Incubator.

(*b*) Additional reagents :—

Congo red papers.
Dimethylamidoazobenzol papers.

I find the best papers are made from smooth filter paper soaked in a watery solution of congo red (grains 3 to 1oz.) and an alcoholic solution of phenolphthaleine (grains 10 to 1oz.). These are then hung up to dry and cut into strips.

Rosalic acid papers (grains 5 to 1oz. alcohol).

These papers are made in same way as congo red papers.

Iodine in solution.

Lugol's solution (liquor iodi compositus).

Potassium Iodide	10 pts.
Iodine	5 „
Distilled Water	100 „

Weak watery solution of Tincture (℥i to ℥i).
Weak alcoholic solution of Tincture (℥i to ℥i).
Gram's Stain.
Cane sugar.
Obermayer's reagent (for Indican) :—

Ferric Chloride in Concentrated HCl (2 pts. to 1,000 pts.).

Nitro-Prusside of Sodium ..
Glacial Acetic Acid .. ..
Liquor Ferri Pechloridi ..
} =required for Acetone and Diacetic Acid.

Methylene Blue Stain.
Fuchsin Stain.
Silver Nitrate Solution.
Corrosive Sublimate Solution (saturated)

Summary of Various Tests.—*HCl* (qualitative). See Chapter XII.

1. Congo red paper, or solution, and gastric filtrate.
   Frankly deep pure blue colour=free HCl.
   Dirty blue or brownish blue=admixture of organic acids.
2. Dimethylamidoazobenzol paper, or solution, and gastric filtrate.
   Pure coral red=free HCl.
   Dirty browny-red=admixture of organic acids.
3. Resorcin-Sugar test (Boas' reagent).

Resorcin	gram 5.
Cane sugar	gram 30.
Alcohol	c.c 100.

   Vermilion red=Free HCl.
4. Phloroglucin-Tannilin test (Gunzburg's reagent).

Phloroglucin	gram 2.
Vanillin	gram 1.
Absolute Alcohol	gram 100.

   The phloroglucin and vanillin are best kept in separate solutions.
   Cherry red=Free HCl.
5. Tropeolin 00 test.
   A few drops of a saturated solution (alcoholic) of tropeolin with a similar quantity of gastric filtrate in porcelain saucer. Changed to red in presence of HCl.

HCl (quantitative). See Chapter XII.

Reagents :—

1% alcoholic solution phenophthaleine.
5% alcoholic solution of dimethylamidoazobenzol.
1% watery solution Sodium Alizarin Sulphonate.
1/10 N solution of Sodium Hydrate (gram 4 to 1,000 c.c. distilled water).

For quantitative estimation of free HCl, total acidity and combined acid. Employed in porcelain saucers and estimated by buret.

Same estimations with Weiss' tube :—

1. Double indicator (Linossier's reagent) :—

Dimethylamidoazobenzol ..	gram .25	=For free HCl and total acidity.
Phenolphthaleine	gram 2	
Alcohol (90%)	gram 100	

2. 1% alcoholic solution of Hæmatoxylin for Combined Acid.

Examination for Organic Acids. See Chapter XII.

1. Lactic Acid.
   Ufflemann's Reagent :—
   2% watery solution of Carbolic Acid 10 c.c.
   Ferric Chloride solution 1 drop.
   Place in test tube and drop gastric filtrate into test fluid. Canary yellow is positive reaction.
   This test is better carried out by means of Strauss' separator to extract lactic acid.

Technique :—

Place 5 c.c. of gastric filtrate in separating funnel (*i.e.*, up to first mark).

On top of this place 20 c.c. of Ether (*i.e.*, up to second mark). Shake vigorously.

Set aside till ether floats to top.

Open stop-cock and run off the 5 c.c. of gastric filtrate.

To the 20 c.c. of ether left add 5 c.c. of distilled water. Shake vigorously.

Set aside till water gravitates to bottom of tube.

Open stop-cock and run off water into clean test tube.

To the watery extract of lactic acid add a couple of drops of a watery solution of ferric chloride (1 in 9).

Canary yellow colour is positive. The intensity of the colour is proportionate to the acid present.

2. Butyric Acid. See Chapter XII.

Gastric filtrate,
Alcohol, of each 6 drops.
Concentrated Sulphuric Acid 2 c.c.

Mixed in test tube. Ethyl Butyrate formed recognised by odour of pine apple.

3. Acetic Acid. See Chapter XII.

Mix 10 c.c. of gastric filtrate with 50 c.c. of Ether. Shake well. Decant ether and evaporate. Dissolve residue in distilled water. Add phenolphthaleine indicator and neutralise with 1/10 N. sodium hydrate solution. Add a few drops of ferric perchloride solution. A red colour is positive of acetic acid.

Blood in Vomitus or gastric content and in fæces. See Chapter XII.

With very minor differences the technique is the same for all of these.

1. Guaiac Test.

(*a*) Place very small pinch of powdered Guaiac in test tube.
Dissolve in 5 c.c. 70% alcohol.
Add equal quantity of active hydrogen peroxide. Mix well.

(*b*) Triturate in mortar small quantity gastric contents or fæces with glacial acetic acid.
Transfer fluid to perfectly clean test tube.
Add an equal quantity of methylated ether.
Invert tube briskly several times.
Set aside till ether separates.
Float ethereal extract with great care on top of ozonized guaiac solution.

A *blue ring* at junction is positive for blood.

2. Aloin Test.

The technique is identical to that of the Guaiac Test, except that for ' very small pinch ' substitute ' large pinch.'

A *red ring* or colour is positive for blood.

3. Teichmann's Hæmatin Test (Strzyzowski's modification).

Place on slide a few drops of gastric filtrate or solution of fæces. Dry over spirit flame. Cover with slip. Insinuate a drop or two of this reagent underneath slip and hold over tiny spirit flame till fluid boils. Replace the reagent which is evaporated from time to time. Reagent :—

Glacial Acetic Acid
Alcohol
Distilled Water .. .. āā 1 c.c.
Hydriodic Acid .. .. m 3.

On cooling black prisms of iodo-hæmatin, seen under microscope, are positive of blood.

4. Benzidin Test.

In test tube dissolve large pinch of benzidin in glacial acetic acid.

In another test tube place small portion of prepared fæces with 2 c.c. distilled water. Boil. Set aside to cool.

In a third test tube place benzidin solution and a few drops of fæcal solution and about 20 drops of Sol. Peroxide of Hydrogen.

The development of a *distinctly blue colour* is positive for blood.

I prefer to carry out the test in a porcelain saucer :—

Distribute a few drops of Benzin and Peroxide solutions over surface of saucer. Flow over this a few drops of fæcal solution. A frankly blue colour, which can be better gauged against the white saucer, is positive. Too delicate to be reliable.

Examination for the Soluble Ferments. See Chapter XII.

Pepsin.

For the practitioner the easiest tests are the indirect ones for the detection of propeptone and peptone :—

1. To 5 c.c. of gastric filtrate add equal quantity of Esbach's reagent in Esbach's tube. Set aside. The amount of precipitate will indicate the activity of the peptic digestion.

2. Incubate 15 grains of hard white of egg with 5 c.c. of gastric filtrate. With normal peptic activity the egg albumin will be completely dissolved in two to four hours.

3. Mett's Test.

Fill capillary glass tube with egg albumin. Plug each end with dough. Put into hot water and quickly boil for five minutes. On removal from water hermetically seal ends with paraffin.

File off two portions 3 m.m. long. Place in undiluted gastric filtrate in incubator. The pepsin activity is estimated by the amount of albumin digested, *i.e.*, the amount of albumin removed from the ends of the tubes. (See text for details.)

4. Ricin Test. (Jacoby-Solms.)

This test is more complicated and will not be much used by the practitioner. Details will be found in Chapter XII.

5. Fuld's Test. (Edistin Test.)

The same remarks apply to this test. (See Chapter XII for details.)

Rennet.

The presence of a milk curdling ferment has been disputed. That it exists, however, is almost certain.

1. Place 10 c.c. of fresh milk in test tube.

Add 5 c.c. of gastric filtrate which has been neutralised by decinormal sodium hydrate solution.

Immerse the tube in water at 37° C. for ten minutes. If rennet be active the milk will have coagulated at the end of this time. No coagulation at the end of thirty minutes shows an absence of rennet.

2. Boas' Test.

This is more complicated. For details see Chapter XII.

Examination of the Fæces. See Chapter XIII.

The examination is usually made after the Schmidt or the Gaultier diets (*q.v.*).

Note microscopic appearances—*e.g.*, quantity, colour, odour, consistency, etc.

Note reaction of fæces: Normally neutral or only slightly acid or alkaline. Liquify stool. Float blue and red litmus paper on top. Note reaction.

Strongly acid = Carbohydrate fermentation.

Strongly alkaline = Albuminous fermentation (ammoniacal decomposition).

Best test is with the fermentation tube of Schmidt and Strasburger (see Chapter XIII.).

Bile Pigment.

Urobilin is normal. Unaltered bile pigment is abnormal.

Make watery solution of fæces. Filter till absolutely clear. Place 5 c.c. in test tube and run on to top very weak alcoholic solution of iodine.

A *green ring* at junction is positive of bile pigment.

Urobilin and Bilirubin easily detected by placing small portion of fæces (solid) in porcelain saucer and covering with saturated watery solution of corrosive sublimate. Place for a short time in the incubator.

A *reddish-pink* colour on surface of fæces indicates *urobilin*.

A *green colour* is positive of *bilirubin*.

Fat in the Fæces.

Microscopically seen as rosettes or spicules or in refractive droplets (see chapter on Pancreatitis).

Albumin in the Fæces.

Make watery solution and filter till clear. Place 5 c.c. in test tube, and acidulate with a few drops of acetic acid. Boil upper portion. Coagulation indicates albumin. Rarely found except in association with pus.

Propeptone and Peptone.

Employ same watery solution as for the albumin test. Place some of the fæcal solution in test tube. Add a little phosotungstic acid. To precipitate which forms add a few drops of weak solution of sodium hydrate and copper sulphate.

A purple-red coloration (Biuret reaction) is positive of the presence of propeptone and peptone.

Microscopic Examination of the Fæces.

Place droplet of fæces on slide. Examine microscopically for meat fibres, starch granules, fat, crystalline salts, hæmatoidin and Charcot-Leyden crystals, epithelium, mucus, pus, blood, tumour tissue, micro-organisms, parasites and ova.

Place drop of fæcal triturate on another slide. Dry in air and pass once or twice through spirit flame.

Stain with Gram's solution. Counter stain with Fuchsin solution.

Normally the general appearance of the field will be *red*. If putrefactive conditions exist the gram-positive organisms will predominate and the field will be *blue*.

Examination of the Motor Power and Absorptive Capacity of the Stomach.

See Chapter XIV.

Motor Power.

If no remnants be recoverable from the stomach in seven hours after the test dinner of Riegel there is adequate motility and no stenosis.

Ewald's Salol Test.

The bladder is emptied and 15 grains of salol given half an hour before food.

The patient is instructed to micturate into numbered glasses every half hour thereafter.

Each sample is tested with ferric perchloride solution for salicylic acid. A burgundy-red colour indicates that the salol has left the stomach. This should be positive in 30 to 70 minutes. If stasis or stenosis exists the reaction may be delayed for two or three hours or indefinitely.

Iodipin Test.

Like salol iodipin is only decomposed in the intestine. Administer 20 grains with test breakfast. When the meal leaves the stomach the iodipin decomposes and iodine is excreted in the saliva. It is detected by dipping starch papers into the saliva and by touching them with a glass rod which has been dipped into HNO_3, which liberates free iodine.

Absorptive Power of Stomach.

Three grains of potassium iodide are administered in capsule. When absorptive power is normal this substance should be detected in the urine and saliva in from six to fifteen minutes. Starch papers are used as above. This is not a very reliable test.

Special Tests for Cancer. See Chapter XIX.

Details of some of these will not be given as they are somewhat complicated and demand the expenditure of a good deal of time.

The following, however, can easily be carried out and may aid diagnosis :—

1. *Wolff-Junghan's Test* (Albumen Test).

This test is best employed to help the differentiation of simple from malignant achylia.

In the absence of HCl no appreciable amount of soluble albumen is formed from the food. If, then, an appreciable amount of albumen is detected in the diluted gastric filtrate it is supposed to be derived from the secretion of the neoplasm (cancer juice) and is positive of malignant disease.

Technique :—

Ewald's test breakfast is given, and, when extracted, tested for occult blood as the smallest contamination of blood would give a precipitate of albumen and vitiate the test.

Take six test tubes and place measured and equal quantities in each of them of the gastric filtrate diluted, as follows :—

Float 1 c.c. of this reagent on the top of the gastric filtrate. A fine pipette should be employed to introduce the reagent so as to avoid intermingling of the fluids :—

Phosphotungstic Acid	0.3 parts.
Hydrochloric Acid	1 part.
Alcohol (90%)	20 parts.
Distilled Water	up to 200 parts.

A precipitate of albumen at the junction of the two fluids is a positive reaction if it occurs in certain dilutions of gastric filtrate. The significance of the reactions is tabulated below :—

1	2	3	4	5	6	Test Tube.
1 in 10	1 in 20	1 in 40	1 in 80	1 in 100	1 in 200	Gastric Filtrate.
W.M.	W.M.	W.M.	M.	T.	F.T.	Normal.
M.	T.	O.	O.	O.	O.	Benign Achylia.
W.M.	W.M.	W.M.	W.M.	W.M.	M.	Malignant Achylia.

W.M.=well marked. F.T.=faint trace.
M.=marked. O.=no precipitate.
T.=trace.

2. *Glycl-Tryptophan Test.*
   Rather complicated. See text, Chapter XIX.

3. *Salomon's Test.*
   Similar in principle to the Wolff-Junghan's test. See text, Chapter XIX.

4. *Gluszinski's Test.*
   This test aims at differentiating a simple ulcer at the pylorus from malignant degeneration of the ulcer.

   Technique :—

   The stomach contents are extracted and tested three times in one day :—

   1. The contents of the fasting stomach.
   2. The gastric contents after a breakfast of egg albumen.
   3. The gastric contents after a test dinner (meat and potatoes).

   If in any one of these examinations a marked hypochlorhydria, or a total absence of HCl is found, malignant degeneration of the ulcer may be suspected.

   The test is unreliable for primary carcinoma, and where ulcer is present it cannot differentiate cancer from chronic atrophic gastritis.

5. *Abderhalden's Test.*
   This is a serum test dependent on a general blood reaction, which is probably only found in advanced cancer. It is therefore not useful in aiding the diagnosis of early carcinoma.

6. *Albumose in Urine.*
   Albumose is not normally found in the urine. But in carcinoma it has been found in about 50% of all cases. Its habitual presence in the urine is suggestive of carcinoma.

Pancreatitis. See Chapter XXIV.

(*a*) *Acute.*

1. *Loewi's Eye Test.*
   Drop into one eye, at short intervals, a few drops of 1 in 1,000 solution of adrenalin.
   Mydriasis is a positive reaction for pancreatitis.

2. *Occult Blood in the Fæces.*
   Positive in almost every case of acute pancreatitis.

3. *Diastase in the Urine.*
   An excess of diastase in the urine is suggestive of acute pancreatitis.

(*b*) *Chronic.*

1. *Macroscopic Appearances of the Stools.*
   The motions are bulky, fatty, and pale. These features are due to failure of pancreatic digestion, enhanced by a diminution or absence of bile in the intestine.

2. *Steatorrhœa.*

By this is meant the evacuation of liquid fat in the fæces.

Split fats are defective in amount.

3. *Creatorrhœa* (*Azotorrhœa*).

By these terms are meant the evacuation of unchanged proteins in the fæces.

Both steatorrhœa and azotorrhœa may be recognised by the microscope.

Fat may occur in refractive globules or in crystalline formation (rosettes and stellate crystals).

Proteins, unchanged, may be recognised by the retention of muscle fibre of its striæ and by the passage through the gut of unchanged stained nuclei.

4. *Test for Pancreatic Sufficiency or Insufficiency.*

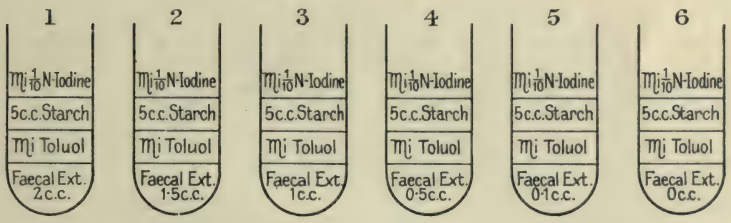
In health the pancreas secretes more than is required for the complete digestion of an ordinary meal. A fæcal extract should, therefore, be capable of digesting a further amount of added starch.

Technique.

1. Make, with pestle and mortar, a 10% solution of the fæces in distilled water. Allow it to settle for two hours.

2. Filter repeatedly till a perfectly clear solution be obtained.

3. To six test tubes add :—



Of the clear fæcal extract 2 c.c., 1.5 c.c., 1 c.c., 0.5 c.c., 0.1 c.c. and 0 c.c. are added to each tube.

A starch solution (1%, *i.e.*, 4½ grains to the ounce of water) is freshly prepared by just bringing the water to the boil. When it has cooled to 100° F, 5 c.c. are added to each tube and shaken well with the fæcal extract.

The drop of toluol is to inhibit any fermentation of the starch by the action of micro-organisms.

Each tube is plugged with cotton wool and incubated for 24 hours, at 37° C.

When removed from incubator add to each tube about 5 c.c. of distilled water, and shake well.

Then add 1 to 2 drops of a decinormal solution of iodine. Note colour developed in each tube.

Blue colour (or blue-black colour)=Undigested starch (dextrin).
Burgundy colour=Partially digested starch (erythrodextrin).
No change in colour=Well-digested starch (achroödextrin).

The dilution in fourth tube is so attenuated that it normally can only partially digest the added starch. Burgundy colour (erythrodextrin) is normal in it.

The first three tubes should remain colourless, because in each of these the pancreatic secretion should be abundant enough to completely digest the starch.

The sixth tube, with no fæcal extract, is used as a control. It stains a deep blue or blue-black colour.

5. *Einhorn's Duodenal Tube.*

Einhorn has shown that the direct introduction of his duodenal tube the pancreatic secretion may be extracted from the duodenum and examined. The ferments will be found normal in activity, defective, or completely absent.

INDEX.

Abdomen, 61
—— Anatomical Considerations, 6
—— Enlargements of, 62—64
—— Fat in wall of, 62
—— Free fluid in, 63
—— Gaseous distension of, 63
—— Physical Examination of, 61-68
—— Regions of, diagrammatic representation, 71
—— Topographical relations of, 69
—— Tumours of, 64, 67
—— Viscera of, 69, 70
Abderhalden's Test for Cancer, 256
Absorption from Stomach, 18, 156
—— from Bowel, 24
—— —— —— of fat, 24
—— —— —— of water, 25
—— —— —— of Saline, 25
—— —— of Nutrient enemata, 25
Abnormalities of Œsophagus, 50-52
Acetic Acid, tests for, 126-127
—— —— fermentation, 21
Acetonæmia, 168, 266
Achlorhydria, 289
Achylia Gastrica (simple or benign), 289
—— —— Diagnosis of, 292
—— —— Diet in, 296
—— —— Etiology of, 290
—— —— Effect of Salt on, 16
—— —— Evacuation of Chyme in, 19
—— —— Relationship to Atrophic Gastritis, 292
—— —— —— to Pernicious Anæmia, 291
—— —— —— to Malignant Disease, 292
—— —— Symptoms of, 293
—— —— Treatment of, 295
—— —— Nervosa, 290
Acidity, investigation of Causes of, 35
Aerophagy, 315
Age, influence of on Gastric disease, 30
Air Swallowing (see aerophagy), 315
Albumen Test for Cancer of the Stomach (Wolff-Junghan's), 252-253
Albumose in urine, in Cancer of the Stomach, 257
Aloin Test for Blood, 130
Amylaceous (Amyl-) Dyspepsia, 15, 142
Amylase, 22
Amylopsin, 22
Amyxorrhœa Gastrica, 298
Anæmia, in Digestive Diseases, 35, 345
—— Simple and pernicious, 36
—— (Chlorosis), relationship to Ulceration, 199
—— in Carcinoma of Stomach, 242
Anamnesis, 26-43
Anginoid pain in peptic ulcer, 206
Anorexia in Carcinoma of Stomach, 242
—— Nervosa, 329
Antacids in gastric ulceration, 222
—— —— —— various formulæ for, 223
Antipepsin, 201
Appendicitis, 23, 340
—— *Acute*, 340
—— *Chronic*, 341
—— —— Differential Diagnosis of, 341
—— —— and Duodenal Ulcer, 341
—— Relationship of Gastric Symptoms to, 23
Arterio-Sclerosis, in peptic ulcer, 199
Ascaris Lumbricoides, in gastric contents, 141
—— —— in fæces, 154
Ascites, 63-66

Atony of Stomach, 300-301
Atrophy of Salivary Glands, 48
Atropine, in Gastric Ulcer, 223
—— in Spasmodic Contraction of Stomach, 272
Atwater and Wood, Food Tables of, 163
Auscultation of Abdomen, 68
Auscultatory Percussion, 68, 84
—— —— Technique, with diagram, 85
Auto-intoxication from Digestive tract, 24
—— in Cirrhosis of Liver, 24
—— in Chronic Renal Disease, 24
—— in Ptomaine Poisoning, 24

Bad Taste in mouth, 41
Barclay, Radiographic Studies of Alimentary Tract, 23
Belching of gas, 34
Belladonna, in Gastric Ulcer, 223
—— in Spasmodic Contraction of Stomach, 272
Benzidin Test for Blood, 131
Biliary Secretion, 21
—— —— Amount of, 21
—— —— Antiseptic Action of, 22
—— —— Characters of, 21
—— —— Functions of, 21-22
Bilirubin, in fæces, 149, 150
Biuret Reaction, 151
Blood, in Vomitus and Gastric Content, 127-128
—— in Vomitus and Gastric Content, conditions in which found, 127
—— Occult, 128, 129
—— in Vomitus, Differential diagnosis, 128
—— —— and fæces, Tests for, 129-133
Boas' Test Breakfast, 110
—— Stomach Tube, 111
—— Test for Rennet, 137
—— —— —— Free HCl, 117
Boas-Oppler Bacillus, 140, 141
Bowels, Bulky Stools, Significance of, 37
—— Colour of Stool, 37
—— Condition of in Gastro-intestinal disorders, 36
—— Constipation of, 37
Bowels, Diarrhœa of, 37, 364, *seq.*
—— Red Blood in, 38
Bulimia (ox-hunger), 327
—— Symptoms and Treatment, 328
Butyric Acid, Fermentation, 21
—— —— Test for, 126

Calorie, Large and Small, 158
Cancer of Cardia, 259
—— of Œsophagus, 58
—— —— Diagnosis and Treatment of, 59-60
—— of Pancreas, 336
—— of Pylorus, 257
—— —— Special features of, 257-258
—— —— Treatment of, 259-263
—— of Stomach, 234-263
—— —— Anæmia in, 36, 242
—— —— Anorexia in, 242
—— —— Associated Changes in Gastric Mucosa, 239
—— —— Constipation in, 244
—— —— Constitutional Disturbance in, 240-263
—— —— Hæmatemesis in, 246
—— —— Leucocytosis in, 243
—— —— Microscopic characters of Vomitus in, 246-248
—— —— Pain in, 244-245
—— —— Prognosis in, 258
—— —— Pyrexia in, 243
—— —— Relationship of indurated ulcer to, 231
—— —— Situations of new growth 239
—— —— Symptoms and signs of, 240-263
—— —— Treatment of, 260
—— —— Urine in, 243
—— —— Varieties of Neoplasm, 237-238
—— —— Vomiting in, 245
Caput Medusæ, 62
Carbohydrates, 19
—— Influence of HCl on, 19
—— —— of Saliva on, 15
Cardioptosis (see Gastroptosis), 273
Cardiac Disease and Dyspepsia, 343
Cardiospasm, 57, 312
—— Etiology of, 312
—— Symptoms and Signs of, 313

Cardio-vascular derangements in Digestive Diseases, 40
Catarrh, Gastric (see Gastritis), 174
Chewing the Cud (merycismus), 316
Cholelithiasis (Gall Stones), 337
Cholecystitis, 339
—— Differential Diagnosis of, 340
—— Jaundice in, 338
—— and Pancreatitis, 339
Chyme, 24
—— Characters of in Stomach, 15
—— —— of in Bowel, 24
—— Rate of movement through tract, 24
Clapotage (see Gastric succussion), 83
Classification of Diseases of the Stomach, 171
Coloptosia (see Gastroptosis), 273
Combined Hydrochloric Acid, Estimation of, 121
Combined Hydrochloric Acid, Sjöqvist Test for in Achylia, 122
Constipation, Chronic, 352
—— —— Abdominal Massage in, 335
—— —— Atonic, 352
—— —— Müller's Exercises in, 365
—— —— Smoking in, 363
—— —— Spastic, 352
—— —— Special Diet and Regimen in, 361
—— —— Treatment, Medicinal of, 357-360
Congo-Red papers, 117
—— Test for Free HCl, 116
Creatorrhœa (see Pancreatitis), 334
Cynorexia (Bulimia), 327

Debio's Method of Distending the Stomach, 88
Dextrose, 23
Diarrhœa, 354
—— in Achylia, 367
—— in fermentation and putrefaction in bowel, 369
—— in functional disorders, 368
—— in Gastroenterostomy, 367
—— of Ingestion, 368
—— Infantile, 369
—— Nervous, 368
—— in Organic Disorders, 365
Diarrhœa, Reflex, 368
—— Treatment of various types of, 366-370
—— in Ulceration of Bowel, 370
—— in Uræmia, 367
Diastase in Urine (see Pancreatitis), 335
Diets, 158
—— in Achylia Gastrica, 296
—— in Atony, Chronic Gastric, 305
—— in Chronic Constipation.
—— in Diarrhœa
—— in Gastritis, *Acute*, 179
—— —— *Chronic*, 190
—— —— —— (Einhorn's), 191
—— in Gastric Carcinoma, 260-261
—— —— Ulcer, 215
Diet Tables, 163-166
Dietetic Principles, 158
Dietl's Crisis (see Movable Kidney), 78
Digestion, Physiology and Chemistry of, 14
Digestion, Physiology and Chemistry of, general summary of, 14-25
Dilatation of Stomach, True obstructive, 264
Dilatation of Stomach, True obstructive, causation of, 264
Dilatation of Stomach, True obstructive, X-ray diagnosis of, 51, 52, 96
Dilatation of Stomach, Acute, 307
—— —— Idiopathic Atonic, 311
Dimethylamidoazobenzol test for free HCl, 117
—— test papers, 117
Diverticula of Œsophagus, 50
—— —— varieties of, 50-51
—— —— Diagnosis and Treatment of, 51, 52
Duodenal Cap, 93
—— —— Ulcer, 196
Dynamic Disorders of Stomach, 281
Dyspepsia, *Acute* (see Acute Gastritis), 174
—— *Chronic* (see Chronic Gastritis) 182
—— of Pulmonary Disorders, 342
—— Anæmia, 345
—— Bronchitis (Chronic), 343
—— of Cardiac Disease, 343

Dyspepsia of Diabetes Mellitus, 345
—— of Nephritis (chronic), 344
—— of Phthisis, 342

Ectasia Ventriculi (see Gastric Dilatation), 311
Edistin Test (Fuld's Reaction) for Pepsin, 136
Eggs, Constituents of, 161
—— Food value of, 161
Einhorn's Diet in Chronic Gastritis, 191
Emaciation in Digestive Affections, 35
Enemata, Rectal, 25, 167
—— Nutrient, 167
—— Simple, 167
Enterokinase, 22-23
—— Action of, 22-23
Enteroptosia, 64, 272
Erepsin, 23
Ewald's Stomach Tube, 110
—— Test Breakfast, 110
—— Test for Gastric Motor Power, 155
Examination of patient, 26
Extraction *versus* Expression of Test Meal, 112

Fæces, Composition of, 24
—— Examination, importance of, 25
—— Albumin in, 150
—— Bacteria in, 145, 153
—— Bile pigment (urobilin) in, 149
—— Bilirubin in, 149-150
—— Blood in, 145
—— Calcium Carbonate and Oxolates in, 152
—— Chemical examination of, 148
—— Consistency of, 146
—— Fat in, 150
—— Fermentation Tube (Strassburger and Schmidt) method of use, 149
—— Hæmatoidin Crystals in, 152
—— Macroscopic appearances of, 144-147
—— Microscopic appearances of, 151-152
—— —— Illustrations of, 150, 152, 153
Fæces, Mucus in, 147
—— Odour of, 146
—— Parasites in, 154
—— Peptone and Propetone in, 151
—— Preparation of by Special Diets, 143
—— Pus in, 148
—— Reaction of, 148
—— Starch in, 152
—— Undigested Food in, 147
—— Weight of, 24, 144
Family History, influence on Stomach Diseases, 31
Faintness in Digestive derangements, 35
Fat in Gastric Contents, 142
Ferments, Soluble, 134
—— Pepsinogen, Pepsin, Rennet, 134-138
Fermentation in Stomach, 20
—— Acetic Acid, 21
—— Butyric Acid, 21
—— Gas Formation in, 20
—— Gastric Retention, 21
—— Yeast in, 20
Flatulence, in various disorders, 34
Floating Kidney, 75-76
—— —— Etiology of, 77
Flushing (Facial) in Digestive Derangements, 40
Foods (see Dietetics), 158
—— Cereals and Vegetables, 164
—— Composition of Common Foods, 163
—— Farm Products, 164
—— Fruits, Nuts, Sugars, 165
—— Importance of preparation of, 14
—— Meat and Game, 163
—— Soups, Tea, Coffee, 165
—— Values of, 159-162
Foreign Bodies, in Œsophagus, 56
Fractional Gastric Analysis, 144
Fuld's Test for Pepsin, 136

Galactose, 23
Gall Stones, 22, 39, 147, 337
Gastralgia Nervosa, 324
—— —— Symptoms and Treatment of, 325-326
Gastrectasis, 311

Gastric Atony, *Acute*, 301
—— —— —— Treatment of, 301
—— —— *Chronic*, 301
—— —— —— Symptoms of, 304
—— —— —— Treatment of, 304
—— —— —— Diet in, 305-306
Gastric Carcinoma, 234-263
—— —— Different Types of, 237-239
—— —— Etiology of, 235-237
—— —— Free HCl in, 248
—— —— Incidence of, 234
—— —— Lactic Acid in, 249
—— —— Pathology of, 237
—— —— Specific Tests for, 251
—— —— Supra clavicular glands in, 250
—— —— Umbilicus in, 250
—— Crisis, 325
—— Dilatation (see Dilatation of Stomach), 264
—— —— *Acute*, 307
—— —— —— Causation of, 307
—— —— —— Clinical Picture of, 308-310
—— —— —— Treatment of, 309-310
—— —— *Chronic*, 311
—— —— —— Distinction from Gastroptosis, 311
Gastric Evacuation in relation to foods, 166
Gastric Douche, 102
—— —— Fever (see Acute Infectious Gastritis), 182
Gastric Hyperæsthesia, 322
—— Juice, 16
—— —— Action of, 18
—— —— Amount secreted daily, 18
—— —— Characters of, 16
—— —— Hydrochloric Acid of, 16
—— —— Method of Secretion of, 16
—— —— Substances which Stimulate, 18
—— Lavage, 98
—— Powder Insufflator, 103
—— Secretion, Disorders of, 281
—— —— Diminution of, 288
—— —— Excess of, 282
—— Ulcer, 196
Gastric Ulcer, After treatment of, 221
—— —— Chemistry of Gastric Contents in, 211
—— —— Chronic Indurated and Carcinoma, 231
—— —— Classical Symptoms of, 203-211
—— —— Deformities of Stomach after, 230
—— —— Diet in, 217-222
—— —— —— (Lenhartz), 217
—— —— —— (Leube - Ziemssen), 222
—— —— Etiology of, 196
—— —— Evolution and Complications of, 225-232
—— —— Hæmorrhage, 209-210
—— —— Hyperchlorhydria in, 197
—— —— Microbic Infection in, 200
—— —— Pain in, 204-208
—— —— Pathological Anatomy of, 202
—— —— Protective Influences against, 200
—— —— Prognosis of, 212
—— —— Rupture of, 225-229
—— —— Situations of, 200
—— —— Subphrenic Abscess in, 228
—— —— Treatment of medical, 212-224
—— —— —— of surgical, 224
—— —— Vomiting in, 208
Gastritis, 174
—— Acid, of Boas, 187
—— *Acute* Catarrhal, 174
—— —— Infectious, 182
—— —— Suppurative, 180
—— Alcoholic, 184
—— Atrophic, 187
—— *Chronic*, 182
—— —— Catarrhal, 182
—— —— Asthenic, 182
—— Glandularis proliferans (see Acid Gastritis), 187
—— Hypersthenic (see Acid Gastritis), 187
—— Pathology of various types, 188
—— Phlegmonous, 180

Gastritis, Toxic, 181
—— Treatment of varieties of, 177
—— —— of Dietetic, Medicinal, and Climatic, 195
Gastro-intestinal History Case-Sheet (see Anamnesis), 27
Gastro-myxorrhœa, 299
Gastroptosis, 64, 82, 272
—— Anatomical Considerations, 273
—— Definition of, 272
—— Diagrammatic Representation of, 275
—— Etiology of, 273
—— Gastric Chemistry in, 276
—— Physical Signs of, 274
—— Symptoms of, 276
—— Treatment of, 277
—— X-ray Examination in, 275
Gastroscopy, 89
Gastro-succorrhœa, 285
—— Continua Chronica, 286
—— —— Periodica, 286
—— Definition of, 285
—— Differentiation from Hyperchlorhydria, 285
—— In Peptic Ulcer, 211
—— In relationship to Duodenal Ulcer, 286
—— Symptoms of, 286
—— Treatment of, 287
Gaultier's Diet for Examination of fæces, 144
Glands, Supraclavicular in Cancer of Stomach, 250
Glénard's Disease (see Gastroptosis), 64, 272
Globus Hystericus, 56
Glossary of Tests, 371-380
Glossitis, 45, 47
Glycyl-Tryptophan Test for Cancer of Stomach, 253-255
Guaiac Test for Blood, 129
Gullet, Examination of, 52, 53
—— —— of by radiography, 92
Gums, 42
—— In purpura, 42
—— Lead poisoning, 42
Günsburg's Test for free HCl, 118

Hæmatemesis, 209
Hæmorrhage, differential diagnosis of, 128, 209
Hæmorrhage, from Stomach, Treatment of, 213-215
Hæmorrhoids, treatment of, 362
Headache in Gastro-intestinal disorders, 36
Heartburn, 35
Hepatosia (see Gastroptosis), 273
Hertz, Radiographic Observations on Stomach, 8
Heterochylia, 298
Holland, C. Thurstan, on Hour-glass Stomach, 272
Hour-glass Stomach, 230, 270
—— —— Gastric Chemistry in, 271
—— —— Inflation of Stomach in, 271
—— —— Treatment of, 272
—— —— Wolfer's Signs in, 271
—— —— X-ray diagnosis of, 97, 271
Hunger Pain, its significance, 32, 207
Hurst, on Etiology of Gastric Ulcer and Appendicitis, 200
Hydrochloric Acid, of Gastric Juice, 16
—— —— Action of, on Bacteria, 16
—— —— in Carcinoma, 248
—— —— *Free*, in test meals, 116
—— —— —— Various qualitative tests for, 116,122
—— —— —— Quantitative estimation of, 118, 120
—— —— —— In relation to total Acidity, 121
—— —— —— In relation to Peptic Ulcer, 18
—— —— Functions of, 16-17
Hyperæsthesia Gastrica, 322
—— —— Symptoms of, 323
—— —— Treatment of, 323
Hyperchlorhydria, 282
—— Causation of, 282, 283
—— Clinical Features of, 283-285
—— Definition of, 282
—— Test Breakfast in, 283
Hyperchylia (see Gastro-succorrhœa), 285
Hyperorexia, 327
Hypochlorhydria, 288
—— in Myxorrhœa, 299

Hypochlorhydria, in relation to Organic Acids, 117
Hypoptyalism, 48

Indicator for Free and Combined HCl, 119
—— for Total Acidity, 119
Inflation of Stomach, 82, 87
Infusoria, in test meals, 140
Inspection of Abdomen, 61
Intestinal Disorders and Gastric Symptoms, 340
Intestinal Motility, 25
—— —— diminished, 25
—— —— increased, 25
—— —— in relation to Constipation, Diarrhœa, and Auto-intoxication, 25
Intestinal Obstructions, 340
—— —— Sand, 146
Introduction, 1-5
Invertase, 23
Iodipin Test for Gastric Motor Power, 156
Iodo-Hæmatin Crystals (Teichmann's), 131

Jacoby-Solms Test for Pepsin, 136

Kidneys, 74
—— Differential Diagnosis from Spleen, 76
—— Examination of, 75
—— Movable, 75
—— —— Association with Gastroptosis, 77
—— —— Symptoms and Signs of, 77-78
—— —— Treatment of, 78
Klemperer's Test for Motor Power of Stomach, 156
Kuttner's Extractor for Test Meals, 113

Lactic Acid, Tests for, 124-125
—— —— —— for, fallacies of, 126
—— —— Significance of, 125-126
—— —— Strauss' Separating Tube for, 125
—— —— Uffleman's Test for, 125
Lactic Acid Bacillus (Oppler-Boas), 140-141
Lactic Acid Bacillus (Oppler-Boas), in Gastric Contents, 140
Lactic Acid Bacillus (Oppler-Boas), in Fæces, 154
Lactic Acid Bacillus (Oppler-Boas), in Cancer of Stomach, 249
Lactic Acid Bacillus (Oppler-Boas), conditions in which found, 126
Lactase, 23
Lactose, 23
Lavage, Gastric, 98
—— —— Antiseptics for, 100
—— —— Conditions Contra indicating, 101
—— —— Friedlieb's Apparatus for, 100
—— —— in Chronic Gastritis, 192
—— —— Technique of, 99
Lenhartz Diet, in Gastric Ulcer, 217
—— —— Author's Modification of, 220

Leptothrix Buccalis, 139
Leube-Riegel Test Dinner, 110
Leube-Ziemssen Cure for Peptic Ulcer, 222
Lipase, 18, 22
—— Action of, 18
Liver, 69
—— Examination of, 71
Loewi's Eye Test in Pancreatitis, 333
Lycorexia (see Bulimia), 327

Malingering and Hæmatemesis, 127
Maltase, 24
Mastication, importance of, 15
Mayo, Wm., on Etiology of Carcinoma of Stomach, 236
Meat Fibres in Gastric Contents, 142

Megalogastria, 311
Melœna, 127, 145
Meteorism, 63
Mett's Test for Pepsin, 135
Milk, Constituents of, 161
—— Food Value of, 161
Motions, Characters of, 36, 39, 144
—— Sieving of, for recovery of Gall Stones, 40
Motor Disorders of Stomach, 300
Motor Insufficiency of Stomach, 300
Motor Insufficiency of Stomach, of 1st degree, 300

Motor Insufficiency of Stomach, of 2nd degree, 311
Motor Power of Stomach, 165
—— —— —— in Cancer, 239
—— —— —— Ewald's Test for, 155
—— —— —— Iodipin Test for, 156
—— —— —— Klemperer's Test for, 156
—— —— —— Riegel's Test Dinner for, 155
Moulds, in Gastric Contents, 141
Mucus in Motions, 39, 147, 148
—— —— Significance of, 39, 147, 148
Mucus of Stomach, 20
—— —— as Contamination, 20
—— —— Determination of origin of, 20
—— —— Diminution of (see Amyxorrhœa), 298
—— —— Excess of (see Myxorrhœa), 299
—— —— As free Secretion, 20
—— —— As protective of mucosa, 200-201
Moynihan, on Etiology of Gastric Carcinoma, 236
Myasthenia Gastrica, 200
—— —— Causation of, 301
Myxorrhœa Gastrica, 299

Neoplasms of Stomach, 233-263
—— —— Simple, 233
—— —— Malignant, 233
Nephritis and Dyspeptic Symptoms, 344
Nephroptosia (see Gastroptosis), 273
Nervous Dyspepsia, 318
—— —— Definition of, 318
—— —— Etiology of, 319
—— —— Treatment of, 320
Neubauer and Fischer Test for Gastric Cancer, 253
Neurasthenia Gastrica (see Nervous dyspepsia), 318
Normal Saline Enemata, 25, 167

Occult Blood, 128, 129, 333
Occupation, influence of, on Gastric Disease, 40
Œsophageal Bougie, 53
Œsophagoscope, 53
Œsophagitis, 55
Œsophagus, 49
—— Abnormalities of, 50
—— Anatomical relations of, 49
—— Description of, 49
—— Examination of, 52, 55
—— Foreign Bodies in, 56
—— Skiagram of, 94
—— Spasm of, 57
—— Stricture of (Simple and Malignant), 57-60
—— Ulceration of, 56
—— X-ray Examination of, 92
Olive Oil, in Peptic Ulcer, 223
Oppler-Boas Bacillus, 140, 141, 247
Oral Cavity, 44
—— —— Diseases of, 44
—— —— —— Stomatitis, 44-45
—— —— —— Glossitis, 45
—— —— —— Treatment of, 45
—— —— Cleansing of, 46
Oral Sepsis, 15, 42
Organic Acids in Stomach Contents, 124, 127
Over-eating, ill effects of, 31
Oxyuris Vermicularis in Gastric Contents, 141

Pain, Gastric, 32
—— —— in Cancer, 244-245
—— —— Causation of in Duodenal and Gastric Ulcer, 204
—— —— Influence of Posture on, 34, 207
—— —— Influence of Food and Alkalies on, 32
—— —— Importance of Study of, in Gastric Disease, 32
Palpation of Abdomen, 65
Pancreas, 79-80, 331
—— Calculi of, 336
—— Carcinoma of, 336
—— Cysts of, 335
—— Diseases of, 331
Pancreatic Efficiency, Test for, 334, 335
Pancreatic Fistula, 22
Pancreatic Secretion, 22
—— —— Characters of, 22
—— —— Stimulation of by Intestinal Secretion, 23
—— —— Trypsogen of, 22

Pancreatitis, *Acute Hæmorrhagic*, 332
—— —— —— Fæces in, 333
—— —— —— Laparotomy in, 333
—— —— —— Loewi's Eye Test in, 333
—— —— —— Symptoms of, 332
—— —— —— Urine in, 333
—— *Chronic*, 333
—— —— Cholelithiasis and Cholecystitis in, 337, 339
—— —— Creatorrhœa in, 334
—— —— Diastase in Urine in, 335
—— —— Einhorn's Duodenal Tube in, 336
—— —— Steatorrhœa in, 334
—— —— Sugar Tolerance in, 335
Parasites in Fæces, 39
Parorexia, 329
Pawlow on Gastric Secretion, 17
Pepsin, 134
—— Edistin Test (Fuld's Reaction), for, 136
—— Mett's Test for, 135
—— Ricin Test (Jacoby-Solms) test for, 136
Pepsinogen, 18
Peptic Ulcer (see Gastric Ulcer), 196
Peptone and Propeptone, tests for, 134
Percussion of Abdomen, 67
—— of Stomach, 83
Perforation of Stomach, 225
—— —— Perigastric adhesions after, 229
—— —— into Peritoneum, 226-227
—— —— into various organs, 227
—— —— Subphrenic Abscess in, 228, 229
Perigastritis, 229-230
Peristaltic Restlessness of Kussmaul (see Tormina Ventriculi), 81, 315
Pernicious Anæmia, 36
—— —— and Achylia, 291
Phalangeal Joints, thickening of in Auto-intoxication, 26
Pharyngitis, 45
Pharynx, 45
Phloroglucin-Vanillin Test for Free HCl, 118
Phthisis and Dyspepsia, 342
Pneumatosis, 316
Polyphagia, 329
Preface, v-vi
Psychic Juice, 162
Ptomaines, 24
Ptalism, 48
Pyloric Stenosis, *Congenital*, 279
—— —— —— Symptoms of, 279
—— —— —— Treatment of, 280
—— —— *Simple*, 63, 264
—— —— —— Auto-intoxication in, 266
—— —— —— Gastric Chemistry in, 269
—— —— —— Gastric Tetany in, 266
—— —— —— Inflation of Stomach in, 267
—— —— —— Clinical Picture of, 264-265
—— —— —— Test Meal in, 268
—— —— —— Visible Peristalsis in, 266
—— —— —— X-ray Examination in, 95, 268
—— —— *Malignant*, 257
—— —— —— Free Hydrochloric Acid in, 257
—— —— —— Sarcinæ in, 258
Pyloric Ulcer, 196
Pylorospasm, 313
—— Symptoms of, 314
—— Treatment of, 315
Pylorus, 23
—— Action of HCl on, 17
—— Automatic Regulation of, 23
—— Barclay's Observations on, 23
—— Glands of, 10
Pyorrhœa Alveolaris, 41
—— —— Detection of, 41
—— —— As pathogenic factor in Peptic Ulcer, 200

Radiography, 90
—— in Cancer of Stomach, 92, 97
—— Opaque Meal in, 91
—— of Œsophagus, 92
—— Preparation of Patient for, 90
—— of Stomach, 92, 97
—— Uses of, 91, 92
Radiotherapy, in Cancer, 97
Rectal Alimentation, 167
—— —— Enemata, 168

Rectal Alimentation, Glucose and Saline Solution in, 168
—— —— Technique of, 169
Recurrent Dyspepsia (see Gastric Ulcer), 196
Regurgitation, Gastric, 317
Rehfuss, on Fractional Gastric Analysis, 114
Reichmann's Disease, 211, 285
Rennin, 18
Rennet, 137
—— Tests for, 137, 138
Resorcin-Sugar Test (Boas) for free HCl, 117
Ricin Test (Jacoby-Solms) for Pepsin, 136
Rosalic Acid, 104
—— —— Test papers for Saliva, 104

Saliva, 15, 104
—— Chemical Examination of, 104, 105
—— Collection of for Chemical Examination, 104, 105
—— Digestion by, 15
—— Diminution of Secretion (hyptotyalism), 48
—— Suppression of Secretion (Xerostomia), 48
—— Hydrolysing Action, 15
—— Increase of Secretion (Ptyalism), 48
—— Reaction of, 15, 104
—— Secretion of, 15
—— —— psychic influence on, 48
Salivary Glands of Dog, 16
—— —— of Man, 47
—— —— Atrophy of, 48
—— —— Secretion, Disorders of, Treatment, 48
Salomon's Test for Cancer of Stomach, 255
Salt, influence of, on HCl Secretion, 16
—— —— on Gastro-succorrhœa, 16
—— —— on Hyperchlorhydria, 16
Sarcinæ Ventriculi, 21, 140
—— —— In Simple Stenosis of Pylorus, 258
—— —— In Malignant Stomach, 258
Schmidt Diet for Examination of the fæces, 143
Scybalæ, 146
Sea Sickness, 351
Secretin, 17
Secretogen, 17
Sensory Disorders of Stomach, 318
Sepsis, Oral, 15
—— Gastro-intestinal, 24
Sex, influence of on Gastric Disease, 30
Silver Nitrate, in treatment of Peptic Ulcer, 224
Sitisphobia, 329
Skin Affections, in digestive derangements, 26, 40
Skiagrams of Stomach, 94, 95
Social Habits and Conditions, influence of, 30
Spasm of Cardia, 312
—— of Pylorus, 313
Spectra of Oxyhæmaglobin and derivitives, 133
Spectroscopic Analysis for Blood, 132
Splanchnoptosia, 64, 272, 273
Spleen, 73
—— Examination of, 73, 74
—— Differential diagnosis from Kidney, 76
Starch Granules in Gastric Contents, 142
Steapsin, 22
Steatorrhœa (see Pancreatitis), 150, 334
Stenosis of Pylorus, *Simple*, prognosis, 258, 264
—— —— Malignant, prognosis, 258
Stimulants, effect of on digestion, 31
Stomach, 6
—— Absorption from, 18, 156
—— Abscess of, 180
—— Adenocarcinoma of, 237
—— Anatomy of, 6
—— Arteries and Veins of, 11, 13
—— Cancer of, 233-251
—— Classification of Diseases of, 171, 172
—— Colloid Cancer of, 238
—— Diagrams of Vascular Supply of, 12
—— Diagrams of Coats of, 9

Stomach, Deformities and Displacements of, 264
—— Dilatation of, 311
—— Diseases of, *organic*, 171
—— —— of, *functional*, 281
—— Distension by water (Dehio's Method), 88
—— Dynamic Disorders of, 281
—— Examination of by Clinical Methods, 81
—— Glands of, 10, 11
—— Inflation of, 87, 88
—— Innervation of, 13
—— Location of Neoplasms of, 239
—— Lymphoid Cells of, 10
—— Motor Power of, 154
—— Mucous Membrane of, 9, 10
—— Mammillæ of Mucous Membrane, 10
—— Mechanical Action of Food on, 18
—— Medullary or Encephaloid Cancer of, 238
—— Motor function Disorders of, 300
—— Movements of, 14
—— Musculature of, 9
—— Perforation of, 225-229
—— Peritoneum of, 9
—— Position of, 6
—— —— when *empty*, 8
—— —— when *half full*, 8
—— —— when *full*, 8
—— Post-mortem appearance of mucosa, 9
—— Relations of, 7
—— Rugæ of, 7
—— Schirrus of, 238
—— Structure of, 9
—— Succussion of, 82
—— Transillumination of, 89
—— Time occupied in emptying, 19
—— Tubes, 111
—— Ulceration of, 196-225
Stomachic Symptoms in disease of other organs, 361
Stomatitis, 44
—— Aphthous, 44
—— Gangrenous, 45
—— Ulcerative, 44
—— Treatment of, 46
Strauss' Test for Lactic Acid, 124, 125
Stricture of Œsophagus, 57-60
Subphrenic Abscess, 228
Succus Entericus, 23
—— —— Characters and functions of, 23
Succussion, Gastric, 82
Supersecretion, Gastric, 285
Syncope, in Hæmatemesis, 215

Tea, effects of abuse of on digestion, 31
Teeth, importance of preservation of, 41, 46
Teichmann's Hæmatin Test for Blood, 131
Test Meals, 109
—— —— Breakfast, 111
—— —— Dinner, 110
—— —— Examination of, 114
—— —— Extractors of, 113
—— —— Free HCl in, qualitative Test for, 116, 118
—— —— Macroscopic appearances of, 114, 116
—— —— Sham, 163
—— —— Microscopic Examination of, 138
—— —— Supper, 110
—— —— Technique of administration and removal, 109-112
Tests, Chemical, Glossary of, 371-380
Tetany, Gastric, 266
Thrush, 45
Tobacco, effects of on digestive functions, 31
Tongue, 42
—— Dry, significance of, 42
—— Cleaning of, 46
—— Coating of in disease, 45
—— As index of digestive derangement, 42
Tonsils, 46
Tormina Ventriculi (see peristaltic unrest), 313
Total Acidity, Estimation of, 120
Transillumination of Stomach, 89
Traube's Space, 84
Tropeolin 00 Test for free HCl, 118

Trypsin, 22
Trypsinogen, 22
Tumours of Stomach (see Cancer of Stomach), 233-263

Ulceration of Stomach (see Gastric Ulcer), 196
—— —— Chronic Indurated, 231
—— —— Post Mortem, 197
Ulceration of Duodenum, 196
—— of Pylorus, 196
—— of Œsophagus, 56
Ufflemann's Test for Lactic Acid, 125
Umbilicus, in Cancer of Stomach, 250
Urine, in Cancer of Stomach, 243, 257
Urobilin in fæces, 149, 150

Vasomotor disturbances in digestive derangements, 35
Vegetable Cells in Gastric Content, 142
Vertigo in digestive derangements, 35
Visceroptosia (see Gastroptosis), 64, 272
Visible Peristalsis of Stomach, 63, 81
—— —— of Bowel, 63
Vitamins, 165
Vomiting as a Symptom, 347
—— Bilious, 348
—— Nervous, 348
Vomiting, in Organic Diseases of Stomach, 319
—— of Pregnancy, 349
—— Persistent, in intestinal obstruction, 348
—— Treatment of, 350
Vomitus, 105
—— Bacteriological Examination of, 108
—— in Cancer of Stomach, 246, 248
—— Macroscopic Examination of, 105-108
—— Microscopic Examination of, 108

Water, action of, on digestion, 19
—— Absorption of, from intestine, 25
—— Evacuation of, from Stomach, 19
Weiss' Tube for estimation of free and combined HCl and total Acidity, 122-124
Wolfer's Signs in Hour-glass Stomach, 271
Wolff-Junghans' Albumen Test for Cancer of Stomach, 252-253
Worms in Motions, 39

Xerostomia, 48
X-rays in Gastric Diagnosis, 89, 90

Yeast, in Test Meals, 140
—— in Cancer of Stomach, 247

Press of The Birmingham Printers, Ltd., Hill Street, Birmingham.

171209

Author Maclennan, William and Craig, J.S.

Title A manual of the diseases of the stomach.

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